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GASTROENTEROLOGY

ABSTRACTS AND CITATIONS

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PREFACE

GASTROENTEROLOGY ABSTRACTS and CITATIONS is a publication of the National Institute of Arthritis, Metabolism and Digestive Diseases. This specialized information medium has been initiated to fill an existing great need in the field of gastroenterology and to assist the Institute in meeting its obligations to foster and support laboratory and clinical research into the nature, causes, and therapy of diseases of the gastrointestinal tract. Publication GASTROENTEROLOGY ABSTRACTS and CITATIONS makes available citations of all current papers relevant to this field from medical journals published throughout the world. Approximately one-third of the citations dealing with the major aspects of gastroenterology have accompanying abstracts.

The issuing of GASTROENTEROLOGY ABSTRACTS and CITATIONS under the auspices of the National Institute of Arthritis, Metabolism, and Digestive Diseases will provide a much needed current awareness tool to scientists and will facilitate greater integration of research and clinical efforts in this field. The number and great diversity of publications in the area of gastroenterology makes it imperative that an appropriate service be available to investigators and practitioners so that they may be apprised of progress with a minimum of delay. Our aim is to provide the readers with a readily systematized compilation of current published work. The publication will provide the greatest usefulness if these interested investigators will contribute their ideas and comments for consideration whenever possible.

GASTROENTEROLOGY ABSTRACTS and CITATIONS is published monthly and includes citations and abstracts from the biomedical world literature as they are currently received. In addition, yearly cumulated subject and author indexes are published.

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LANGUAGE ABBREVIATIONS

Afr.	Afrikaans	It.	Italian
Ar.	Arabic	Jpn.	Japanese
Bul.	Bulgarian	Kor.	Korean
Ch.	Chinese	Latv.	Latvian
Cz.	Czech	Lith.	Lithuanian
Dan.	Danish	Nor.	Norwegian
Dut.	Dutch	Pol.	Polish
E.	English	Por.	Portuguese
Eston.	Estonian	Rum.	Rumanian
Fin.	Finnish	Rus.	Russian
Fl.	Flemish	Ser.	Serbo-Croatian
Fr.	French	Sl.	Slovak
Ger.	German	Sp.	Spanish
Gr.	Greek	Sw.	Swedish
Heb.	Hebrew	Th.	Thai
Hun.	Hungarian	Turk.	Turkish
Ic.	Icelandic	Uk.	Ukrainian
In.	Indonesian	Viet.	Vietnamese

ABBREVIATIONS USED IN ABSTRACTS

ACTH	adrenocorticotropic hormone	mm	millimeter(s)
ADP	adenosine diphosphate	mOsm	milliosmolar; milliosmeter(s)
AMP	adenosine monophosphate	msec	millisecond(s)
amp	ampere	mV	millivolt(s)
ATP	adenosine triphosphate	min	minute(s)
Å	angstrom	MTD	maximum tolerated dose
BSP	sulfobromophthalein	NAD	nicotanamide adenine dinucleotide
C	degrees centigrade	NADH	reduced nicotanamide adenine dinucleotide
cal	calories		
cc	cubic centimeter	NADP	nicotanamide adenine dinucleotide phosphate
cm	centimeter(s)		
dl	deciliter	NADPH	reduced nicotanamide adenine dinucleotide phosphate
DNA	deoxyribonucleic acid		
ED_{50}	median effective dose(s)	ng	nanogram (10^{-9})
EDTA	ethylenediamine tetraacetic acid	nm	nanometer(s)
e.g.	for example	Pa	ambient pressure
EEG	electroencephalogram	PAS	periodic acid-Schiff
ESR	erythrocyte sedimentation rate	pg	picogram (10^{-12})
g	gram(s)	pgEq	picogram equivalent(s)
µg	microgram(s)	p.o.	orally
Hz	Herz	ppb	parts per billion
hr	hour(s)	ppm	parts per million
i.m.	intramuscular	r	Roentgen
i.p.	intraperitoneal	RBC	red blood cells (erythrocytes), red blood count
IU	international unit(s)		
i.v.	intravenous	resp.	respectively
kcal	kilocalorie(s)	RNA	ribonucleic acid
kg	kilogram(s)	s.c.	subcutaneous
K_m	Michaelis constant	sec	second(s)
LD_{50}	median lethal dose(s)	SGOT	serum glutamic-oxalacetic transaminase
LDH	lactic acid dehydrogenase		
m	meter(s)	SGPT	serum glutamic-pyruvic transaminase
M	molar; mole(s)		
mM	millimolar; millimole(s)	U	unit(s)
µM	micromolar; micromole(s)	UV	ultraviolet
ma	milliampere(s)	V	volt
mCi, µCi	milli-, microcurie(s)	WBC	white blood cells (leukocytes), white blood count
mEq	milliequivalent(s)		
mg	milligram(s)	yr	year(s)
ml, µl	milli-, microliter(s)		

PRE-CLINICAL SCIENCES

MORPHOLOGY, CELLULAR STRUCTURE AND HISTOCHEMISTRY

0001 CORRELATIVE LIGHT AND ELECTRON MICROSCOPICAL STUDIES ON THE G (GASTRIN) AND D ENDOCRINE CELL TYPES OF THE HUMAN PYLORIC MUCOSA. *(E.)* Canese, M. G.; Bussolati, G. (Fac. Med., Turin Univ., Italy). *Rend. Gastroenterol.* 6(1):12-22, 1974.

The staining characteristics and the ultrastructural morphology of the endocrine cells of the human pyloric mucosa, obtained from 2 stomachs surgically removed because of duodenal ulcer, were correlated on serial sections. With the indirect immunofluorescence procedure numerous gastrin-containing cells were detected in the pyloric glands. Gastrin cells were detected by immunofluorescence in semithin sections from aldehyde-fixed, Epon-araldite embedded tissues. Similar results were obtained using osmium to post-fix the cells. In aldehyde-fixed, osmium post-fixed tissues several cell types could be recognized on electron microscopy, the most prominent in number being the G cell, with typical round, "floccular" granules. Numerous D cells were also observed, while only a few enterochromaffin cells and rare cells with round, small granules (D_1 cells) were seen. In tissues fixed in aldehyde alone, the endocrine granules did not display the typical morphology because of the lack of membrane staining. The endocrine cells selectively stained by immunofluorescence, histological and histochemical methods in light microscopy were traced on the ultrastructural sections. In most instances, the transfer process from light to electron microscopy, and *vice versa*, was successful. The gastrin cells detected by immunofluorescence were easily identifiable in tissues post-fixed in osmium and were characterized in electron microscopy by all the features which are known to be typical of the G cells. The D cells were negative with the immunofluorescence method for gastrin. The argyrophilic Davenport-positive cells and the metachromatic cells positive with the HCl-Toluidine Blue method were identified as corresponding to the D cell type. The discrepancy between these findings and those from other authors is attributed to experimental methodology. The authors conclude that this study leaves no doubt that G cells produce gastrin, but that the function of D cells is still unknown.

0002 ORIGIN, DIFFERENTIATION AND RENEWAL OF THE FOUR MAIN EPITHELIAL CELL TYPES IN THE MOUSE SMALL INTESTINE. I. COLUMNAR CELL. *(E.)* Cheng, H.; Leblond, C. P. (Dep. Anat., McGill Univ., Montreal, Canada). *Am. J. Anat.* 141(4):461-480, 1974.

The distribution and behavior of epithelial columnar cells from the duodenum, jejunum, and ileum of adult Swiss mice sacrificed at various times after single injection or continuous infusion of ^{3}H-thymidine were studied with light and electron microscopy and autoradiography. A single s.c. injection of 1 µCi/g of labeled thymidine to 8 female mice resulted in 96% or more of the labeled cells being free of secretory granules, and thus columnar, 1 hr after injection. These columnar cells accounted for 92.4, 86.0, and 84.1% of the cells in the crypts of the duodenum, jejunum, and ileum, resp. and 97.3, 95.0, and 87.6% of the cells in the villi of the duodenum, jejunum, and ileum, resp. These high percentages indicate the likelihood of columnar cells playing a key role in the renewal of the epithelium in the small intestine. The turnover time of columnar cells, estimated from the results of continuous infusion of labeled thymidine (1.5 µCi/g/day) to male mice sacrificed in groups of 2 at 10 hr, 2 days, and 4 days, was 3.3 days in the duodenum and 3.4 days in the jejunum. The turnover time data indicate that the mitoses of columnar cells produce more cells than required for their own renewal, with some of the mitoses presumably giving rise to cells of other types. Electron microscopic studies of columnar cells at different levels of the epithelium following a single i.p. injection of 2 µCi/g of labeled thymidine to 30 male mice sacrificed from 1-144 hr after injection indicated that columnar cells exhibit local differences. They were classified into 4 subgroups: (I) crypt-base columnar cells, immature proliferative cells occupying the 9 lowest cell positions of the crypt; (II) mid-crypt columnar cells, proliferative rudimentary differentiating cells occupying the next positions up to 19 in the ileum and 27 in the duodenum and jejunum; (III) crypt-top columnar cells, nonproliferating differentiating cells occupying the rest of the crypt; (IV) villus columnar cells, fully differentiated absorptive cells. The migration of labeled columnar cells from crypt to villus was visualized with light-microscopic examination of autoradiographs. In the jejunum, 1 hr after labeled thymidine injection (2 µCi/g), heavily labeled columnar cells were confined to crypt-base and mid-crypt positions. By 6 hr heavily labeled cells appeared in the crypt-top and by 12 hr at the base of some villi, while labeling intensity decreased in the lower portions of the crypts. Thereafter, heavily labeled columnar cells were observed ascending the villus epithelium and eventually reaching the villus tip where they dropped into the lumen.

0003 ORIGIN, DIFFERENTIATION AND RENEWAL OF THE FOUR MAIN EPITHELIAL CELL TYPES IN THE MOUSE SMALL INTESTINE. II. MUCOUS CELLS. *(E.)* Cheng, H. (Dep. Anat., McGill Univ., Montreal, Canada). *Am. J. Anat.* 141(4):481-502, 1974.

The distribution and behavior of eipthelial mucous cells from the duodenum, jejunum, and ileum of adult Swiss mice sacrificed at various times after single injection or continuous infusion of ^{3}H-thymidine were studied with light and electron microscopy and autoradiography. Single s.c. injection of 1 µCi/g of labeled thymidine to 8 female mice resulted in the characterization of 2 types of mucous cells: granular and common. The granular mucous cells differ from the common ones by the presence of small dense granules embedded within the mucous globules. Each subgroup was further subdivided into immature oligomucous cells containing few mucous globules and mature goblet cells. Common and granular oligomucous cells were found exclusively in the crypt, mainly within the lower mid-crypt, whereas the 2 types of goblet cells were present in the upper part

of the crypts and in the lower part of the villi. Only common mucous cells were observed in the upper part of the villi. Electron microscopic autoradiography after single i.p. injection of 2 μCi/g labeled thymidine to 30 male mice sacrificed at time intervals ranging from 1-144 hr demonstrated that as oligomucous cells migrate upwards, they transform into goblet cells which in turn migrate to the villus epithelium. These findings were also observed after continuous infusion of 1.5 μCi/g/day of labeled thymidine to male mice divided into groups of 2, sacrificed after 10 hr, 2 days, and 4 days. The labeling pattern of granular mucous cells was determined after single i.p. injection of labeled thymidine (1 μCi/g) to 45 female mice sacrificed at intervals ranging from 1-336 hr. Granular mucous cell migration was associated with a gradual loss of the characteristic dense granules, so that the granular goblet cells reaching the upper part of the villi became common goblet cells. The goblet cells in the villus epithelium, regardless of their origin, ascended towards the villus tips where they were lost through the extrusion zones. The turnover time of common mucous cells was about 3 days, being somewhat shorter for granular types. Divisions of oligomucous cells accounted for the production of only about half of the mucous cells present. Hence, the other half must have been derived from precursors other than oligomucous cells. Since a few crypt-base columnar cells contained the odd mucous globule, these were suspected as being the precursors of the 2 types of oligomucous cells and, through them, the entire mucous cell population.

0004 ORIGIN, DIFFERENTIATION AND RENEWAL OF THE FOUR MAIN EPITHELIAL CELL TYPES IN THE MOUSE SMALL INTESTINE. III. ENTERO-ENDOCRINE CELLS. *(E.)* Cheng, H.; Leblond, C. P. (Dep. Anat., McGill Univ., Montreal, Canada). *Am. J. Anat.* 141(4):503-520, 1974.

The distribution and behavior of epithelial entero-endocrine cells from the duodenum, jejunum, and ileum of adult Swiss mice sacrificed at various times after single injection or continuous infusion of ^{3}H-thymidine were studied with light and electron microscopy and autoradiography. Although light microscopic examination after s.c. injection of 1 μCi/g of labeled thymidine to 8 female mice did not reveal mitosis in the entero-endocrine cells (iron hematoxylin stained), electron microscopy identified a few cells that contained a small number of characteristic granules, some filament bundles, and many free ribosomes that were in division or were labeled 1 hr after injection. These cells were interpreted as young entero-endocrine cells. They were located in the crypt base and resembled crypt-base columnar cells which perhaps gave rise to them. Cell differentiation was examined by following the fate of the young entero-endocrine cells after a single i.p. injection of 2 μCi/g labeled thymidine to 30 male mice. When s-collidine buffered glutaraldehyde was used for fixation prior to electron microscopic study, all granules in all entero-endocrine cells were spheroidal, and thus their shape could not be used to classify the cells into subgroups. Fixation with glutaraldehyde in phosphate buffer resulted in the observation of both irregular and spheroidal granules. The shape of the granules seemed to be influenced only by the buffer. Within the crypts, these cells acquired a gradually increasing number of granules, while losing the ability to divide. The few granules initially present usually had a particulate content; but, as differentiation proceeded and granules accumulated, their content was mostly dense and homogeneous. The differentiation of precursor cells into mature entero-endocrine cells took about 2 days. In the same manner as columnar and mucous cells, entero-endocrine cells migrated up the crypt, reached the villus, and were lost at the extrusion zone. Infusion of 1.5 μCi/g/day of labeled thymidine to male mice divided into groups of 2 sacrificed after 10 hr, 2 days, and 4 days allowed for turnover time estimates of 3.9 days in the duodenum and 4.0 days in the jejunum.

0005 ORIGIN, DIFFERENTIATION AND RENEWAL OF THE FOUR MAIN EPITHELIAL CELL TYPES IN THE MOUSE SMALL INTESTINE. IV. PANETH CELLS. *(E.)* Cheng, H. (Dep. Anat., McGill Univ., Montreal, Canada). *Am. J. Anat.* 141(4):521-536, 1974.

The distribution and behavior of epithelial Paneth cells from the small intestine of adult Swiss mice sacrificed at various times after single injections of ^{3}H-thymidine were studied with light and electron microscopic autoradiography. Injection (s.c.) of 8 female mice with 1 μCi/g labeled thymidine revealed that Paneth cells make up 3.3% of the crypt epithelial cells in the duodenum, 7.5% of the cells in the jejunum, and 6.6% of the cells in the ileum. The Paneth cell population consisted of 23% young Paneth cells with granules less than 2 μ in diameter, 63% mature Paneth cells with granules 2-3 μ in diameter, and 14% old Paneth cells with granules greater than 3 μ in diameter. Light and electron microscopic autoradiography confirmed that typical Paneth cells are not labeled 1 hr after thymidine injection and, therefore, are not capable of proliferation. Labeled cells with a few small Paneth cell granules were, however, observed 12 hr after injection. Except for the presence of granules, these cells were indistinguishable from the highly proliferative crypt-base columnar cells which are suspected to be the precursors of Paneth cells. Cell differentiation was studied after i.p. injection of 2 μCi/g labeled thymidine to 30 male mice sacrificed at periods of 1-144 hr following injection. Stacks of rough endoplasmic reticulum cisternae appeared as differentiation proceeded, with the granules becoming progressively more numerous and larger and the nucleus acquiring an irregular shape. Labeled mature Paneth cells were observed by 5 days after injection. Unlike other types of small intestinal epithelial cells, Paneth cells did not migrate up to the villus epithelium and were thus not lost through the extrusion zone. Instead, they underwent degeneration in the crypt-base and were eventually phagocytosed by an adjacent crypt-base columnar cell.

0006 THE CELL CYCLE TIME IN THE RAT JEJUNAL MUCOSA. *(E.)* Al-Dewachi, H. S.; Wright, N.

A.; Appleton, D. R.; Watson, A. J. (Dep. Pathol., Univ. Newcastle upon Tyne, England). *Cell Tissue Kinet.* 7(6):587-594, 1974.

The cell cycle time as determined from curves of percent labeled mitoses was studied in different layers of the jejunal crypt mucosa of male albino Wistar rats injected i.p. previously with ^{3}H-thymidine. For the whole crypt, the mean cycle duration was 11.32 hr and the mean duration of the DNA synthetic phase was 6.49 hr. Cells in the bottom 8 layers showed significantly prolonged cell cycle (12.34 to 15.48 hr) and DNA synthetic phase (7.14 to 8.56 hr) durations. The cell cycle parameters in the remaining cell layers did not differ significantly; the mean cell cycle and DNA synthetic phase durations were close to 11 and 6 hr, resp., with a relatively narrow distribution. The results indicate that the fall in proliferative indices seen toward the top cell layers of the crypt is not due to prolongation of the cell cycle duration as previously suggested but is fully consistent with the slow cut-off model.

0007 THE STRUCTURE OF RAT LIVER MITOCHONDRIA: A REEVALUATION. *(E.)* Brandt, J. T.; Martin, A. P.; Lucas, F. V.; Vorbeck, M. L. (Univ. Missouri Sch. Med., Columbia). *Biochem. Biophys. Res. Commun.* 59(3):1097-1103, 1974.

The liver from male Charles River rats was removed after carotid exsanguination and small sections were fixed in 2% glutaraldehyde buffered to pH 7.4 (6-8 hr) and post fixed in osmium tetroxide. The tissue was dehydrated in graded concentrations of ethanol and then embedded in Epon 812. Sections (0.08 μ) were prepared using a Porter-Blum ultramicrotome. The sections were stained with lead hydroxide and uranyl acetate and examined with a Hitachi HS-7 electron microscope. The configurations of the mitochondria observed were simple rod shapes, longer filamentous rods, V-shaped, disk-shaped, and more complex branched, tubular structures. Branching was seen in every cell examined, but branching was not observed in every mitochondrial profile. In any given section, several rod to ovoid mitochondria profiles may be part of the same mitochondrion. The variety of forms seen and the types of interrelationships observed suggest that the structure of the mitochondria may be quite plastic. Because rat liver mitochondria undergoes marked structural changes under varying nutritional states, the organization and control of intracellular structure becomes important, and a model is necessary for the study of these mechanisms and how they may relate to various physiologic and pathologic states. Studies in mitochondrial biogenesis in mammalian system which were based on the premise of a uniform population of mitochondrion relatively free in the cytoplasm should be reassessed.

0008 MINOR SALIVARY GLANDS OF MAN: ENZYME AND MUCOSUBSTANCE HISTOCHEMICAL STUDIES. *(E.)* Harrison, J. D. (King's Coll. Hosp. Dent. Sch., London, England). *Histochem. J.* 6(6):633-647, 1974.

0009 SALIVARY GLANDS OF THE CAT: A HISTOCHEMICAL STUDY. *(E.)* Harrison, J. D. (King's Coll. Hosp. Dent. Sch., London, England). *Histochem. J.* 6(6):649-664, 1974.

0010 HISTOCHEMICAL STUDY OF LIPIDS IN THE SUBMAXILLARY AND SUBLINGUAL GLANDS OF THE GUINEA PIG. *(Fr.)* Gotzos, V.; Musy, J. P. (Inst. Histol. Gen. Embryol., Univ. Fribourg, Switzerland). *Histochemistry* 41(2):145-160, 1974.

0011 ETHIONINE INDUCED DEGENERATION AND REGENERATION IN THE RAT PAROTID GLAND: AN ELECTRON MICROSCOPE STUDY. *(E.)* Leeb, I. J. (Med. Coll. Virginia, Richmond). *Am. J. Anat.* 142(1):29-46, 1975.

0012 INTRODUCTION: THE VILLUS IN CHEMOSTAT MAN. *(E.)* Luckey, T. D. (Univ. Missouri Sch. Med., Columbia). *Am. J. Clin. Nutr.* 27(11):1266-1276, 1974.

0013 ENDOSCOPIC BIOPSIES OF NORMAL DUODENAL MUCOSA. *(E.)* Korn, E. R.; Foroozan, P. (Scripps Clin. Res. Found., La Jolla, Calif.). *Gastrointest Endosc.* 21(2):51-54, 1974.

0014 MORPHOLOGY OF EXTRAGANGLIONIC FLUORESCENT NEURONS IN THE MYENTERIC PLEXUS OF THE SMALL INTESTINE OF THE RAT. *(E.)* Oosaki, T.; Sugai, N. (Fukushima Med. Coll., Japan). *J. Comp. Neurol.* 158(1):109-120, 1974.

0015 HISTOLOGICAL STUDIES OF THE RABBIT CECUM AND COLON. *(It.)* Andreozzi, G.; Cioffi, L. A.; Proto, V. (Dep. Gen. Histol., Univ. Naples, Italy). *Boll. Soc. Ital. Biol. Sper.* 49(21):1247-1252, 1973.

0016 STUDY ON SUBMUCOSAL VEINS OF THE HUMAN ILEOCECAL VALVE. *(Ger.)* de Carvalho, C. A. F.; Costacurta, I.; Furlani, J.; Nogueira, J. O. (Inst. Anat., Univ. Sao Paulo, Brazil). *Anat. Anz.* 135(5):495-503, 1974.

0017 THE FINE STRUCTURE OF THE NEXUS BETWEEN SMOOTH MUSCLE CELLS OF THE TAENIA COLI IN FREEZE-ETCHED REPLICAS. *(Ger.)* Geisweid, G.; Wermbter, G. (Med. Coll., Hanover, Germany). *Cytobiologie* 9(2):121-130, 1974.

0018 DISTRIBUTION AND RELEASE OF GASTRIN IN HUMAN GASTRIC MUCOSA. *(E.)* Tobe, T.; Henmi, K.; Fukuchi, K.; Chen, S. T.; Tachibana, S. (2nd Surg. Dep., Kyoto Univ., Japan). *Acta Histochem. Cytochem.* 7(1):79, 1974.

MORPHOLOGY, CELLULAR STRUCTURE AND HISTOCHEMISTRY

0019 ASSIGNMENT OF THE GENE FOR GALACTOKINASE TO HUMAN CHROMOSOME 17 AND ITS REGIONAL LOCALIZATION TO BAND q21-22. *(E.)* Elsevier, S. M.; Kucherlapati, R. S.; Nichols, E. A.; Creagan, R. P.; Giles, R. E.; Ruddle, F. H.; Willecke, K.; McDougall, J. K. (Dep. Biol., Yale Univ., New Haven, Conn.). *Nature* 251(5476):633-636, 1974.

0020 A METHOD FOR PRECISE LOCATION OF THE MEDIAN FISSURE OF THE LIVER (PRELIMINARY REPORT). *(E.)* Kekis, B. P.; Yiannakakis, A.; Dayadas, J.; Andypas, C.; Astras, G. (Greek Church Hosp., Athens, Greece). *J. Med. Liban.* 27(4):463-466, 1974.

0021 IN SITU LOCALIZATION OF GLOBIN MESSENGER RNA FORMATION. I. DURING MOUSE FETAL LIVER DEVELOPMENT. *(E.)* Harrison, P. R.; Conkie, D.; Affara, N.; Paul, J. (Beatson Inst. Cancer Res., Glasgow, Scotland). *J. Cell Biol.* 63(2):402-413, 1974.

0022 MICROPHOTOMETRY OF RAT LIVER NUCLEOPROTEINS I. DIFFERENCES IN NAPHTOL YELLOW S TOTAL NUCLEOPROTEIN AND FASTGREEN HISTON [SIC!] CONTENT BETWEEN PARENCHYMAL AND LITTORAL CELL NUCLEI. *(E.)* Morselt, A. F. W.; Maarschalkerweerd, V. (Histol. Lab., Univ. Amsterdam, Netherlands). *Histochemistry* 41(2):107-110, 1974.

0023 MICROPHOTOMETRY OF RAT LIVER NUCLEAR PROTEINS II. MICROPHOTOMETRY OF RAT LIVER NUCLEAR PROTEINS AND RNA BEFORE AND AFTER PARTIAL HEPATECTOMY. *(E.)* Morselt, A. F. W.; Frederiks, W. M. (Histol. Lab., Univ. Amsterdam, Netherlands). *Histochemistry* 41(2):111-118, 1974.

0024 MORPHOMETRIC STUDY OF CELL STRUCTURE IN THE RAT LIVER DURING HORMONE INDUCTION. *(Rus.)* Khristoliubova, N. E.; Shilov, A. G.; Kiseleva, E. V.; Salganik, R. I. (Inst. Cytol. Genet., Novosibirsk, USSR). *Tsitologiia* 15(3):254-259, 1973.

0025 ALTERATIONS IN THE FINE STRUCTURE OF HEPATOCYTES IN HYPERTHYROID RATS. *(E.)* Callas, G.; Cannon, M. S. (Univ. Texas Med. Branch, Galveston). *Anat. Rec.* 181(1):71-82, 1974.

0026 STRUCTURE AND FUNCTION OF BILE CANALICULAR MICROFILAMENTS. *(E.)* Phillips, M. J.; Oda, M.; Fisher, M. M.; Jeejeebhoy, K. N. (Dep. Med., Univ. Toronto, Canada). *Am. J. Pathol.* 78(1):13a-14a, 1974.

See also, 0036, 0039, 0082, 0445, 0776.

ABSORPTION—EXCRETION—TRANSPORT

0027 THE ABSORPTION OF ^{125}I-LABELLED IMMUNOGLOBULIN G BY DIFFERENT REGIONS OF THE GUT IN YOUNG RATS. *(E.)* Morris, B.; Morris, R. (Dept. Zool., Univ. Nottingham, England). *J. Physiol. (Lond.)* 241(3):761-770, 1974.

The ability of different regions of the small intestine of young rats to absorb ^{125}I-labeled immunoglobulin G (IgG) and transmit it to the circulation was investigated. ^{125}I-labeled homologous IgG was injected into different regions of the small intestine of albino Wistar rats aged 12, 16, 18, 20, and 22 days. At 12 days the proximal and middle regions of the intestine readily absorbed globulin and transmitted it to the circulation. The distal region of the intestine transmitted little to the circulation at all ages tested. At 16 and 18 days the ability of the middle region to transmit globulin to the circulation had declined significantly; this decline continued so that little globulin was transmitted from this region at 20 and 22 days. The proximal intestine retained the ability to transmit globulin to the circulation in significant amounts up to 20 days. There was a close negative correlation between body weight and total radioactivity of the sera of rats which had received doses of labeled globulin into the proximal and middle regions of the intestine, but no such correlation after injection into the distal intestine, suggesting a restricted throughput of radioactive material by the absorptive cells of the distal small intestine. It is probable that the labeled rat globulin injected into the distal part of the intestine was taken up by the absorptive cells and accumulated in the supranuclear vacuoles, as was polyvinyl pyrrolidone (PVP) in previous experiments. Since there was good correlation between the age of termination of antibody absorption in young rats and the age when PVP uptake ceased, it was suggested that the loss of the ability to take up substances of high molecular weight might be a factor in limiting the period of antibody absorption. Since this change in the distal intestine (that is the replacement of PVP-permeable cells by PVP-impermeable cells) does not relate to the cessation of globulin transmission, its functional significance remains to be determined.

0028 STUDIES IN VIVO AND IN VITRO OF THE TRANSFER OF RAT IgG AND RAT ALBUMEN ACROSS THE INTESTINAL WALLS OF YOUNG RATS. *(E.)* Jones, R. E. (ARC Immunol. Dep. Zool., Univ. Coll. North Wales, Bangor). *Biol. Neonate* 24(3-4):220-229, 1974.

In vivo and *in vitro* studies of the transfer of rat immunoglobulin G (IgG) and rat albumin across the walls of the small intestine were performed. Equal amounts (500 µg) of rat ^{125}I-albumin and ^{131}I-IgG in physiological saline were fed together to Wistar rats at 3, 14, and 24 days of age, followed by sacrifice 3 hr later. The concentration quotients (cpm/ml of serum divided by cpm/ml of fed dose) for labeled albumin and labeled IgG were about the same in the 3-day-old rats and indicated fairly free absorption of the proteins into the circulation. In 14-day-old rats the concentration quotient for the transmission of IgG was about 20 times higher than for rat albumin. In 24-day-old rats insignificant amounts of either protein were found in the sera. Washes taken from the small intestine 30 min after feeding were subjected to centrifugation and gel filtration on Sephadex G-100 and indicated that 64% (measured planimetrically) of the ^{131}I activity in 3-day-old rats was from whole IgG molecules, while only 21% of the ^{125}I counts corresponded to undegraded albumin. Washes from 14-day-old rats indiccated that 78% of the breakdown products were present from ^{125}I-labeled albumin, whereas the bulk of the ^{131}I activity was still associated with substantially undegraded IgG. Proteolysis had proceeded much further in the lumen of the 24-day-old rats, with only 7 and 13% of the total activities corresponding to undegraded albumin and immunoglobulin G, resp. *In vitro* studies with everted sacs prepared from the proximal, mid, and distal third regions of the small intestine of 14- and 23-day-old rats indicated that for both age groups the ^{125}I- and ^{131}I-labeled proteins were transported intact and in considerable quantities across the whole of the small intestine. The transference of both proteins occurred most actively across the mid sections.

0029 EFFECT OF VITAMIN D DEFICIENCY ON AMINOACIDURIA AND INTESTINAL TRANSPORT OF AMINO ACIDS IN THE RAT. *(E.)* Morin, C. L.; Leveille, J.; Ling, V. (St. Justine Hosp., Montreal, Canada). *Can. J. Physiol. Pharmacol.* 52(5):972-977, 1974.

Studies over a 16-wk period to induce hyperaminoaciduria in 120 male weanling Holtzman rats maintained on 2 pelleted semisynthetic vitamin D deficient diets differing only by their calcium content (0.4 and 0.04% by weight) were performed to investigate the possibility of a concomitant defect in intestinal transport of amino acids. Animals with vitamin D deprivation developed progressive hypocalcemia, with the condition being most severe for rats on a low calcium diet. Phosphorus excretion was greater from the 2nd week onwards in animals on vitamin D-deficient low calcium diets than in controls. Vitamin D-deprived animals receiving normal calcium amounts developed higher phosphorus excretions than controls from the 6th week onwards. Despite signs of secondary hyperparathyroidism, generalized hyperaminoaciduria was not observed in any of the groups. Increased urinary excretion of lysine and taurine was observed, however, in rats fed a low calcium diet without vitamin D. The same observation was also made for taurine in rats deprived of vitamin D and on a normal calcium diet as well as for animals fed a low calcium diet with vitamin D. The administration of calcium gluconate (4 mg/8 hr i.p. for 3 days) and vitamin D_2 (10,000 U/day i.m. for 2 consecutive days) to 3 of the rats on low calcium, vitamin D-deficient diets did not result in different intracellular accumulations of L-lysine, cycloleucine, and L-alanine as compared to values measured for the remainder of the group. The experiments thus failed to show any effect of vitamin D deficiency, hypocalcemia, or secondary hyperparathyroidism on the intestinal transport of amino acids.

0030 JEJUNAL ABSORPTION OF AN AMINO ACID MIXTURE SIMULATING CASEIN AND AN ENZYMIC HYDROLYSATE OF CASEIN PREPARED FOR ORAL ADMINISTRATION TO NORMAL ADULTS. *(E.)* Silk, D. B. A.; Clark, M. L.; Marrs, T. C.; Addison, J. M.; Burston, D.; Matthews, D. M.; Clegg, K. M. (St. Bartholomew's Hosp., London, England). *Br. J. Nutr.* 33(1):95-100, 1975.

An intestinal perfusion technique utilizing a double-lumen cuffed tube in the jejunum was used in 6 normal human volunteers to study absorption of 16 amino acids from an amino acid mixture simulating casein and from an enzymatic hydrolysate of casein consisting of 50% oligopeptides and 50% free amino acids. Amino acid absorption was estimated by determining those remaining in the perfusate using ion-exchange chromatography. There was considerable variation in the extent of absorption of individual amino acids from the amino acid solution with methionine, leucine, and isoleucine being well-absorbed and threonine and histidine being extremely poorly absorbed. Amino acids were much more uniformly and extensively absorbed from the casein hydrolysate with phenylalanine, alanine, tyrosine, serine, aspartic acid, threonine and histidine being absorbed to a greater extent than from the amino acid mixture. Total absorption of α-amino nitrogen was 29% greater from the casein hydrolysate (40.3%) than from the equivalent amino acid solution (31.2%). These results indicate that administration of protein hydrolysates rather than amino acid mixtures may be nutritionally preferable in patients with extensive resections or malabsorption.

0031 ACTIVE TRANSPORT OF L-ASCORBIC ACID IN THE HUMAN ILEUM. *(E.)* Stevenson, N. R. (Rutgers Med. Sch., Piscataway, N.J.). *Gastroenterology* 67(5):952-956, 1974.

L-Ascorbic acid was absorbed by an independent Na^+-dependent active transport mechanism in ileal tissue taken from the human intestine. Tissue uptake studies demonstrated absorption against a gradient and a requirement for cellular energy as demonstrated by the large inhibition (86%) observed in the presence of the metabolic inhibitor, 2,4-dinitrophenol. A requirement for sodium was demonstrated in 2 ways. Ouabain, an inhibitor of the Na^+-K^+-dependent ATPase, caused a substantial inhibition (54%) of ascorbic acid accumulation, as did the partial replacement of Na^+ by either K^+ (65%) or $Tris^+$ (71% inhibition). The observation that glucose enhanced ascorbic acid transport (41%), presumably by acting as an energy metabolite, rather than inhibiting it, would appear

to indicate that L-ascorbic acid does not use the monosaccharide transport system. Phlorizin, a competitive inhibitor of monosaccharide transport, added in a concentration sufficient to block essentially all glucose uptake, was observed to cause partial inhibition of ascorbic acid transport (45%). This is attributed to phloretin release by phlorizin hydrolase, and thus provides additional indication of the energy requirement of this transport system. Thus, L-ascorbic acid is absorbed by a Na^+-dependent active transport mechanism in man which is similar to that in the guinea pig.

0032 UPTAKE AND METABOLISM OF PLASMA GLUTAMINE BY THE SMALL INTESTINE. *(E.)* Windmueller, H. G.; Spaeth, A. E. (Natl. Inst. Health, Bethesda, Md.). *J. Biol. Chem.* 249(16):5070-5079, 1974.

Measurements were made of the net uptake of glutamine by isolated perfused rat intestine. Preparations extracted large amounts of glutamine (75 μM/hr) but no other amino acids from a recirculated blood perfusate. With L-[*U*-^{14}C]glutamine, the carbon was partly incorporated into tissue acid-insoluble material (14%) and the rest reappeared with little delay in intestinal venous blood in CO_2 (57%), citrulline (6%), proline (5%), and organic acids (18%), predominantly citric acid and lactic acid. Perfusate glutamine was the source of 32% of the CO_2 produced by the preparation, although the perfusate contained 10 to 15 mM glucose. Most glutamine uptake and metabolism occurred in the small intestinal mucosal cells and, from analyses of labeled tissue metabolites, appeared to proceed *via* glutamic acid. The rapid flow of glutamine carbon through mucosa may be explained in part by the small glutamine pool, only 0.2 μM/g of tissue. Glutamine nitrogen taken up by intestine could be approximately accounted for by the quantities of citrulline (34%), alanine (33%), ammonia (23%), and proline (10%) released back into the perfusate. Glutamine uptake comparable to that found in the isolated organ was observed *in vivo*, from arteriovenous concentration measurements across intestine in fasted dogs, cats, hamsters, monkeys, and conventional and germ-free rats. Twenty-five to 33% of plasma glutamine was extracted in each pass through the tissue. Glutamine uptake persisted in rats during transport of large amounts of glutamic acid from the intestinal lumen into the blood and in rats injected with 6-diazo-5-oxo-L-norleucine, a glutamine analog (65 mg/kg). The results of these studies suggest that glutamine derived from blood is an important respiratory substrate for cells in the small intestinal mucosa and that intestine is a major site for metabolism of glutamine released into the circulation by other tissues.

0033 CHARACTERISTICS OF SODIUM FLUX FROM SEROSA TO MUCOSA IN RAT ILEUM. *(E.)* Desjeux, J. F.; Tai, Y. H.; Curran, P. F. (Yale Univ. Sch. Med., New Haven, Conn.). *J. Gen. Physiol.* 64(3):274-292, 1974.

Sodium flux from serosa to mucosa in rabbit ileum *in vitro* was studied as a function of applied electrical potential at equal sodium concentrations in the bathing solutions. Pieces of distal ileum, stripped of their serosal and muscle layers, were mounted as flat sheets between two Lucite half-chambers which were connected to jacketed reservoirs of bathing solution maintained at 37 C. The bathing solutions were connected *via* agar bridges to calomel electrodes for measurement of potential difference and to silver-silver chloride electrodes for passing current through the tissue. Isotopes and pharmacological agents when used were added to the bathing solutions immediately after mounting the tissue. The sodium flux from serosa to mucosa appeared to involve two pathways: a diffusional flux through a paracellular shunt pathway and a flux that was independent of applied potential and presumably involved a transcellular pathway. The latter pathway comprised approximately 25% of the sodium flux from serosa to mucosa in Ringer's solution containing 10 mM glucose and 25 mM bicarbonate. This presumably transcellular pathway was stimulated significantly by theophylline, unaffected by removal of glucose or the addition of ouabain, and reduced to negligible values of sodium flux by anoxia, 2,4-dinitrophenol, and replacement of all chloride and bicarbonate by isethionate. This component of sodium flux from serosa to mucosa thus demonstrated a number of characteristics consistent with involvement in a specific secretory process mediating an electrically neutral secretory transport of sodium plus anion from serosa to mucosa. Theophylline, in addition to stimulating this process, significantly reduced the permeability of the paracellular shunt pathway to sodium.

0034 BLOCKAGE OF CATION PERMEABILITY ACROSS THE TIGHT JUNCTIONS OF GALLBLADDER AND OTHER LEAKY EPITHELIA. *(E.)* Moreno, J. H. (Univ. California Med. Cent., Los Angeles). *Nature* 251(5471): 151, 1974.

The protonated form of the organic base 2,4,6-triaminopyrimidine (TAP) was used to reversibly and specifically block cation permeation across the tight junction route (between cells) of the frog gallbladder and several other leaky epithelia. Transepithelial electrical potential differences and conductances were measured across flat sheets of bullfrog gallbladder mounted between 2 well-stirred chambers at 23 C, and the rate of fluid transport was measured in sac preparations of bullfrog gallbladder by a gravimetric procedure. Addition of TAP was to both the mucosal and serosal sides of the preparation and invariably increased the membrane resistance and decreased the sodium to chloride permeability ratio due to a specific decrease in sodium conductance. The pyrimidine did not affect chloride conductance, indicating that sodium and chloride migrate through separate pathways and that the chloride pathway is a relatively small parallel leakage or free solution shunt. In 5 other leaky epithelia (6 rabbit gallbladders, 2 Necturus gallbladders, 2 rabbit small intestines, 2 bullfrog small intestines, and 2 bullfrog choroid plexuses), TAP had qualitatively the same effect as in frog gallbladder. The rate of isotonic solution transport, which measures and is caused by active transport of sodium from the mucosal to the serosal solutions, in the absence of sodium bicarbonate in the bathing solutions was 3.9 μl/hr/

cm^2 in 6 frog gallbladders and, after addition of 10 mM TAP, 3.8 µl/hr/cm^2. When 7 mM $NaHCO_3$ (which stimulates active transport) was included in the bathing solutions, the rate of transport increased to 9.8 in controls and to 9.9 µl/hr/cm^2 in the presence of 10 mM TAP. Thus, TAP at a concentration which blocks 80% of the transepithelial sodium conductance does not seem to interfere with active sodium transport or to affect bicarbonate stimulation of transport. Gallbladder TAP in the frog appears to act specifically in the cation-selective ion channels of the tight junction responsible for the passive sodium permeability without affecting either the small chloride leakage current or the active sodium transport.

0035 INHIBITION OF IRON ABSORPTION IN MAN BY TETRACYCLINE. *(Ger.)* Heinrich, H. C.; Oppitz, K. H.; Gabbe, E. E. (Univ. Hosp. Eppendorf, Univ. Hamburg, Germany). *Klin. Wochenschr.* 52(10): 493-498, 1974.

The inhibition of iron absorption by tetracycline was studied in humans. A therapeutic 500 mg dose of tetracycline (Hostacyclin), combined with ^{59}Fe-labeled bivalent ion cations in the form of a 50 mg therapeutic dose of iron sulfate in aqueous solution, was administered orally to subjects with normal and depleted iron reserves. The patients were fasted for 10 hr before and 2 hr after administration. The whole body retention of absorbed ^{59}Fe was measured within the 4π counting geometry of a large-volume radioactivity detector by means of a liquid organic scintillator. When administered simultaneously with iron, tetracycline reduced iron absorption from 7.9 to 1.6%, corresponding to an inhibition of 78% in subjects with normal iron reserves, and from 15 to 5.4%, corresponding to an inhibition of 65%, in subjects with depleted iron reserves. When tetracycline and iron were administered simultaneously but in separate capsules, tetracycline inhibited iron absorption by 32 in subjects with normal iron reserves, and by 40 in subjects with depleted iron reserves. The findings indicate that tetracycline is a potent inhibitor of iron absorption due to the formation of stable, sparingly absorbable iron-tetracycline chelates through inhibition or total suppression of the biosynthesis of carrier proteins responsible for the control of iron absorption. During tetracycline therapy, iron and other bivalent metallic cations should never be administered concomitantly. However, an interval of 3 hr is sufficient to avoid drug interaction with iron.

0036 ELECTRICAL IMPEDANCE, ULTRASTRUCTURE AND ION TRANSPORT IN FOETAL GASTRIC MUCOSA. *(E.)* Wright, G. H. (Dep. Physiol., Bedford Coll., London, England). *J. Physiol. (Lond.)* 242(3):661-672, 1974.

0037 THE K^+-STIMULATED ATPase SYSTEM OF MICROSOMAL MEMBRANES FROM GASTRIC OXYNTIC CELLS. *(E.)* Forte, J. G.; Ganser, A. L.; Tanisawa, A. S. (Dep. Physiol. Anat., Univ. California, Berkeley). *Ann. NY Acad. Sci.* 242:255-267, 1974.

0038 INVESTIGATIONS INTO THE EFFECT OF PRENYLAMINE ON ABSORPTION IN HUMAN SMALL INTESTINE. *(Ger.)* Gottesburen, H.; Raida, H.; Riecken, E. O. (Med. Clin., Univ. Marburg/Lahn, Germany). *Dtsch. Med. Wochenschr.* 99(42):2104-2105, 1974.

0039 EFFECT OF LANTHANUM AND SITS (4-ACETAMIDO-4'-ISOTHIOCYANOSTILBENE-2,2'-DISULFONIC ACID) ON IONIC SELECTIVITY OF THE PROXIMAL INTESTINE IN Rana esculenta. *(It.)* Schettino, T.; Curci, S.; Lippe, C. (Inst. Gen. Physiol., Univ. Bari, Italy). *Boll. Soc. Ital. Biol. Sper.* 49(18 bis):no. 74, 1973.

0040 STUDIES ON DRUG NONEQUIVALENCE. I. THERMODYNAMIC PARAMETERS AND INTESTINAL ABSORPTION OF POLYMORPHS OF GLUTATHIONE. *(E.)* Kuroda, K.; Nomura, T. (Kobe Univ. Hosp., Japan). *Kobe J. Med. Sci.* 20(2):47-54, 1974.

0041 THE EFFECT OF PINEALECTOMY ON INTESTINAL ABSORPTION OF GLYCINE IN THE GROWING RAT. *(Fr.)* Peres, G. E.; Vareilles, F. (Univ. Med., Clermont-Ferrand, France). *C. R. Soc. Biol. (Paris)* 168(1):54-58, 1974.

0042 INTESTINAL ABSORPTION OF TRYPTOPHAN STEREOISOMERS AND ANALOGS. *(E.)* Bosin, T. R.; Hathaway, D. R.; Maickel, R. P. (Med. Sci. Program, Indiana Univ., Bloomington). *Arch. Int. Pharmacodyn.* 212(1):32-35, 1974.

0043 STUDIES ON THE UPTAKE OF GLYCYL-L-LEUCINE BY STRIPS OF MONKEY SMALL INTESTINE. *(E.)* Das, M.; Radhakrishnan, A. N. (Christ. Med. Coll. Hosp., Vellore, India). *Indian J. Biochem. Biophys.* 11(1):12-16, 1974.

0044 INTESTINAL TRANSFER OF BIVALENT CATIONS: EFFECTS ON THE ABSORPTION OF ^{45}Ca OF INCREASING DOSES OF L-ARABINOSE INGESTED SIMULTANEOUSLY WITH THIS MINERAL. *(Fr.)* Fournier, P.; Dupuis, Y. (Fac. Sci. Pharm. Biol., Chatenay-Malabry, France). *C. R. Acad. Sci. [D] (Paris)* 278(24):3107-3110, 1974.

0045 ROLE OF GASTRIC ACID IN THE UTILIZATION OF DIETARY CALCIUM BY THE RAT. *(E.)* Mahoney, A. W.; Hendricks, D. G. (Nutr. Food Sci., Utah State Univ., Logan). *Nutr. Metab.* 16(6):375-382, 1974.

0046 STIMULATION OF CALCIUM ABSORPTION AND APPARENT INCREASED INTESTINAL 1,25-DIHYDROXYCHOLECALCIFEROL IN RATS TREATED WITH LOW DOSES OF ETHANE-1-HYDROXY-1,1-DIPHOSPHONATE. *(E.)* Guilland, D.; Trechsel, U.; Bonjour, J. P.; Fleisch, H. (Dep. Pathophysiol., Univ. Bern, Switzerland). *Clin. Sci. Mol. Med.* 48(2):157-160, 1975.

ABSORPTION–EXCRETION–TRANSPORT

0047 FACULTATIVE CONTROL OF INTESTINAL CALCIUM ABSORPTION. *(E.)* Cramer, C. F. (Dep. Physiol., Univ. British Columbia, Vancouver, Canada). *Rend. Gastroenterol.* 6(2):132-138, 1974.

0048 RELATIONSHIP BETWEEN CADMIUM AND IRON ABSORPTION. *(E.)* Hamilton, D. L.; Valberg, L. S. (Dep. Med., Queen's Univ., Kingston, Canada). *Am. J. Physiol.* 227(5):1033-1037, 1974.

0049 IRON ABSORPTION AND TOLERANCE TEST IN IRON DEFICIENCY. *(Sp.)* Sottano de Russo, E. Y.; Marsano, O.; Arroyo, N. (Fac. Med. Sci., Univ. Natl. Cordoba, Argentina). *Rev. Assoc. Bioquim. Argent.* 38(209/210):284-287, 1973.

0050 INTERACTION OF TRANSFERRIN WITH IRON-BINDING SITES ON RAT INTESTINAL EPITHELIAL CELL PLASMA MEMBRANES. *(E.)* Evans, G. W.; Grace, C. I. (Agric. Res. Serv., U.S. Dep. Agric., Grand Forks, N.D.). *Proc. Soc. Exp. Biol. Med.* 147(3): 687-689, 1974.

0051 EFFECT OF DIETARY IRON MANIPULATION AND PHENOBARBITONE TREATMENT ON IN VIVO INTESTINAL ABSORPTION OF IRON IN MICE WITH SEX-LINKED ANEMIA. *(E.)* Edwards, J. A.; Hoke, J. E. (Dep. Med., State Univ. New York, Buffalo). *Am. J. Clin. Nutr.* 28(2):140-145, 1975.

0052 DEPENDENCE OF INCREASED IRON ABSORPTION BY IRON-DEFICIENT RATS ON AN ELUTABLE COMPONENT OF JEJUNAL MUCOSA. *(E.)* Huebers, H.; Huebers, E.; Rummel, W. (Inst. Pharmacol. Toxicol., Univ. Saarland, Homburg/Saar, Germany). *Hoppe Seylers Z. Physiol. Chem.* 355(9):1159-1161, 1974.

0053 EFFECT OF GLUCAGON ON RADIOCALCIUM EXCRETION BY THE COLON IN RATS AFTER INTRACARDIAL ADMINISTRATION OF ^{45}Ca. *(E.)* Bogdal, J.; Jedrychowski, A.; Tarnawski, A.; Dura, K.; Marszalek, Z. (Med. Acad. Krakow, Poland). *Can. J. Physiol. Pharmacol.* 52(6): 1230-1232, 1974.

0054 THE EFFECT OF ALUMINUM CHLORIDE ON ^{45}Ca FLUXES IN ISOLATED LONGITUDINAL SMOOTH MUSCLE FROM RAT COLON. *(E.)* Hava, M.; Hurwitz, A. (Univ. Kansas Med. Cent., Kansas City). *Arch. Int. Pharmacodyn.* 212(1):24-31, 1974.

0055 ABSORPTION OF IRON. HUMORAL FACTORS AND HEPATIC DEPOSITION. *(Sp.)* Sottano de Russo, E. Y.; Itoiz, J. E.; Arroyo, N. (Fac. Med. Sci., Univ. Natl. Cordoba, Argentina). *Rev. Assoc. Bioquim. Argent.* 38(209/210):288-291, 1973.

0056 TRANSFERRIN AND IRON UPTAKE BY THE LIVER IN THE RAT. *(E.)* Gardiner, M. E.; Morgan, E. H. (Dep. Physiol., Univ. West. Australia, Nedlands). *Aust. J. Exp. Biol. Med. Sci.* 52(5):723-736, 1974.

0057 THE PASSIVE TRANSPORT OF POTASSIUM IN RAT LIVER CELLS. *(E.)* Mazet, J. L.; Claret, M.; Claret, B. (Lab. Comp. Physiol., Natl. Sci. Res. Cent., Orsay, France). *J. Membr. Biol.* 18(3/4):335-350, 1974.

0058 INHIBITORY EFFECT OF PROSTAGLANDINS ON ISOSMOTIC FLUID TRANSPORT BY RABBIT GALLBLADDER IN VITRO, AND ITS MODIFICATION BY BLOCKADE OF ENDOGENOUS PGE-BIOSYNTHESIS WITH INDOMETHACIN. *(E.)* Leyssac, P. P.; Bukhave, K.; Frederiksen, O. (Univ. Inst. Exp. Med., Copenhagen, Denmark). *Acta Physiol. Scand.* 92(4):496-507, 1974.

0059 UDP GLUCURONYLTRANSFERASE AND PHENOLSULFOTRANSFERASE FROM RAT LIVER IN VIVO AND IN VITRO. CHARACTERIZATION OF CONJUGATION AND BILIARY EXCRETION OF HARMOL IN VIVO AND IN THE PERFUSED LIVER. *(E.)* Mulder, G. J.; Hayen-Keulemans, K.; Sluiter, N. E. (Dep. Pharmacol., State Univ. Groningen, Netherlands). *Biochem. Pharmacol.* 24(1):103-107, 1974.

0060 PARTICIPATION OF Na,K-DEPENDENT ATPase IN THE ACTION MECHANISM OF OXYTOCIN IN ISOTONIC FLUID TRANSPORT IN THE GALLBLADDER EPITHELIUM. *(Rus.)* Iaremenko, M. S.; Butusova, I. A.; Kharlamova, O. N. (A. A. Bohomolets Inst. Physiol., Kiev, USSR). *Fiziol. Zh. SSSR* 60(10):1592-1596, 1974.

See also, 0012, 0149, 0322.

MOTILITY

0061 INTERACTION OF PENTAGASTRIN AND THE OCTAPEPTIDE OF CHOLECYSTOKININ ON THE HUMAN LOWER OESOPHAGEAL SPHINCTER. *(E.)* Sturdevant, R. A. L.; Kun, T. (VA Wadsworth Hosp., Los Angeles, Calif.). *Gut* 15(9):700-702, 1974.

Seven adult male volunteers without clinical evidence of gastric or esophageal disease were studied with motility tube assemblies to determine if infusion of the octapeptide of cholecystokinin inhibits the response of the lower esophageal sphincter to pentagas-

trin. After positioning of the motility tube assembly, an i.v. infusion of either 154 mM sodium chloride or cholecystokinin (40 ng/kg/hr) was begun, with lower esophageal sphincter pressure being recorded for 10 min. Pentagastrin was then given through a 3-way stopcock attached to the infusion in doses of 0.05, 0.1, 0.2, 0.4, and 0.6 µg/kg (over 30 sec at 10 min intervals in random order). Lower esophageal sphincter pressure was recorded as the mean of end-inspiratory and end-expiratory pressures in mm Hg greater than intragastric pressure. The octapeptide of cholecystokinin infusion did not significantly decrease basal lower esophageal sphincter pressure but partially inhibited the response to pentagastrin. The inhibition was significant for 0.1, 0.2 and 0.4 µg/kg doses, with the mean peak lower esophageal sphincter pressure in mm Hg decreasing from about 30 to 20, 25 to 20, and 35 to 25, resp. The data did not fit Michaelis-Menten kinetics sufficiently well to determine if the inhibition was competitive or not. In contrast, bolus injections of octapeptide of cholecystokinin (5 ng/kg and 20 ng/kg) to 3 of the same subjects lowered the lower esophageal sphincter pressure from 15.3 mm Hg before injection to 9.5 and 2.2 mm Hg, resp. after injections. This action was inhibited by a background infusion of 0.5 µg/kg/hr of pentagastrin such that the lower esophageal sphincter pressures after the bolus injections of 5 ng/kg and 20 ng/kg octapeptide of cholecystokinin were 17 and 7.3 mm Hg. The results confirm the view that gastrin and cholecystokinin have pharmacological effects on lower esophageal sphincter pressure, though their roles, if any, in its physiological control are not established. If gastrin is not involved in regulation of basal lower esophageal sphincter pressure, then the decrease in lower esophageal sphincter pressure produced by bolus injection of octapeptide of cholecystokinin cannot be explained by inhibition at a postulated cholecystokinin-gastrin receptor. This action could be due to direct activation of an inhibitory receptor rather than to blocking of a stimulatory receptor.

0062 MECHANISM OF THE INHIBITORY ACTIONS OF SOME SPASMOGENS ON THE BRADYKININ RELAXATION OF THE GUINEA-PIG ILEUM. *(E.)* Hall, D. W. R.; Bonta, I. L.; Verhoef, E. R. (Med. Fac., Erasmus Univ., Rotterdam, Netherlands). *Eur. J. Pharmacol.* 25(2):308-315, 1974.

The effects of various intestinal spasmogens on bradykinin-induced relaxation of guinea-pig ileum were studied in isolated preparations. Increasing doses of histamine (20-200 ng/ml) and eledoisin (10-200 ng/ml) to concentrations exceeding their maximal effect showed increasing abilities to inhibit bradykinin (2 ng/ml)-induced small bowel relaxation, although high concentrations were less effective than previously reported effects of high concentrations of acetylcholine. Angiotensin (4-100 ng/ml) treatment showed no such dose-response relationship although low non-spasmogenic doses in the presence of acetylcholine-induced contraction did reduce bradykinin relaxation. Both morphine (10 ng/ml) and atropine (1 ng/ml) blocked 1,1-dimethyl-4-phenylpiperazinium iodide-induced (1 µg/ml) small bowel contractions by 77.4% and 45.4% resp. Both substances also significantly reduced angiotensin-induced contraction and potentiated bradykinin-induced relaxation in the presence of angiotensin. Morphine had no effect whereas atropine (0.5 ng/ml) partially blocked acetylcholine-induced small bowel contraction. Neither morphine nor atropine had any effect on bradykinin reduction of its own relaxation response on acetylcholine-treated ileum. Thus, the magnitude of the bradykinin relaxation on guinea-pig ileum depends on the manner of stimulation rather than on the state of contraction and is sensitive to regulation by parasympathomimetic agents. Angiotensin reduces bradykinin-induced relaxation *via* a mechanism which involves its indirect parasympathomimetic effects. However, bradykinin reduction of its own relaxing effect is most probably by direct action rather than *via* a parasympathetic mechanism.

0063 INTERACTION BETWEEN SEROTONIN AND MORPHINE IN THE GUINEA-PIG ILEUM. *(E.)* Gintzler, A. R.; Musacchio, J. M. (New York Univ. Med. Ctr., N.Y.). *J. Pharmacol. Exp. Ther.* 189(2):484-492, 1974.

The effect of 5-hydroxytryptamine on morphine-induced inhibition of electrically induced concentrations of terminal guinea-pig ileum was studied *in vitro*. 5-Hydroxytryptamine (2×10^{-8} to 2×10^{-7}M) produced a 2- to 3-fold increase in the degree of inhibition of ileum contraction by morphine (2×10^{-8}M). Conversely, pretreatment with small concentrations of morphine (5×10^{-9} to 10^{-8}M) potentiated the inhibitory response of ileum to 5-hydroxytryptamine (4×10^{-7}M) by about 3-fold. The potentiating effect of 5-hydroxytryptamine was also observed for nalorphine (4-fold) and methadone (2-fold) but not for norepinephrine (10^{-8}M). Norepinephrine had no effect on morphine-induced inhibition of contraction. Lysergic acid diethylamide (0.1 µg/ml) antagonized the inhibitory effects of 5-hydroxytryptamine and produced a 2-fold potentiation of the morphine effect. These results suggest that the interaction between morphine and 5-hydroxytryptamine is specific and that 5-hydroxytryptamine may mediate the effects of morphine on the small intestine.

0064 THE EFFECTS OF HUMAN- AND ANIMAL-PATHOGENIC COLI STRAINS ON THE ISOLATED INTESTINE OF RABBIT. *(Ger.)* Metz, H.; Bonu, D.; Ohgke, H. (Max von Pettenkofer Inst. Hyg. Med. Microbiol., Univ. Munich, Germany). *Zentralbl. Bakteriol.* [*Orig. A*] 226(2):215-223, 1974.

The effect of human- and animal-pathogenic coli strains on the motility of the isolated, sacciform prepared ileum of rabbit was studied. The addition of enteropathogenic coli bacteria induced characteristic changes in the rhythms of the contractions of the ileum in Krebs-Henseleit solution as recorded on kymograph drum. This effect was found among O types of *E. coli* pathogenic for infants (*E. coli* O 55 and O 111 strains) in 139 out of 276 experiments, and among O types of swine enteritis or swine edema disease (O 8, O 138, O 149, and O 139 strains) in 59 out of 64 experiments. Heat treatment at 100 C and aging of the cultures did not influence the results. Sodium chloride suspensions of bacteria cultured on solid medium had a significantly lower effect. Non-

pathogenic coli strains did not influence the motility of the ileum preparations. The disturbance in the motility rhythm of the ileum was induced by the lipopolysaccharide-free supernatant of ultrasonic homogenates rather than by the lipopolysaccharides of the bacteria. The results indicate the possible existence of a protein-like enterotoxin causing colienteritis in infants and young pigs.

0065 THE ACTION MECHANISM OF RESTRAINT ON GASTRIC EMPTYING IN THE RAT. III. ROLE OF THE ADRENAL GLANDS. *(Fr.)* Dumas, J.; Peres, G. (Lab. Gen. Compar. Physiol., Univ. Lyon, France). *C. R. Soc. Biol. (Paris)* 168(1):47-50, 1974.

0066 EFFECT OF PENTAGASTRIN ON THE LOWER ESOPHAGEAL SPHINCTER: AN ANIMAL EXPERIMENT. *(Ger.)* Kunath, U.; Hollevoet, J. (Surg. Cent., Johann Wolfgang Goethe Univ., Frankfurt/Main, Germany). *Langenbecks Arch. Chir.* 336(3):235-246, 1974.

0067 EFFECT OF TEMPERATURE ON ELECTRICAL ACTIVITY IN THE STOMACH AND DUODENUM OF ANESTHETIZED RATS. *(Fr.)* Medici, A.; Morelle, O.; Thouvenot, J. (Lab. Physiol., UER Med., Univ. Tours, France). *C. R. Soc. Biol. (Paris)* 168(2/3):326-330, 1974.

0068 EFFECT OF ACTH (CORTROSYN-DEPOT) ON ELECTROMYOGRAMS ON STOMACH AND SMALL INTESTINES. *(E.)* Milenov, K. T.; Nikolov, N. A.; Kasakov, L. N. (Bulgarian Acad. Sci., Sofia). *Dokl. Acad. Nauk (Bulg.)* 27(8):1149-1152, 1974.

0069 DEVELOPMENT OF TOLERANCE TO THE STIMULATORY EFFECT OF MORPHINE IN DOG INTESTINE. *(E.)* Burks, T. F.; Jaquette, D. L.; Grubb, M. N. (Univ. Texas Med. Sch., Houston). *Eur. J. Pharmacol.* 25(2):302-307, 1974.

0070 SITES OF ACUTE MORPHINE TOLERANCE IN INTESTINE. *(E.)* Burks, T. F.; Grubb, M. N. (Univ. Texas Med. Sch., Houston). *J. Pharmacol. Exp. Ther.* 191(3):518-526, 1974.

0071 SOME EFFECTS OF DIPYRIDAMOLE, HEXOBENDINE AND LIDOFLAZINE IN INHIBITORY PROCESSES IN RABBIT DUODENUM. *(E.)* Hulme, M. E.; Weston, A. H. (Dep. Pharmacol., Univ. Manchester, England). *Br. J. Pharmacol.* 50(4):609-611, 1974.

0072 STRUCTURE-ACTIVITY RELATIONSHIP OF NATURAL AND SEMI-SYNTHETIC GENINS AND GLYCOSIDES AS INVESTIGATED ON THE SMOOTH MUSCLE OF THE GUINEA-PIG ILEUM. *(E.)* Haustein, K. O.; Megges, R.; Hauptmann, J. (Med. Acad. Erfurt, Germany). *Pharmacology* 10(2):65-75, 1973.

0073 MODULATING ROLE OF PROSTAGLANDINS IN CONTRACTIONS OF THE GUINEA-PIG ILEUM. *(E.)* Kadlec, O.; Masek, K.; Seferna, I. (Inst. Pharmacol., Prague, Czechoslovakia). *Br. J. Pharmacol.* 51(4): 565-570, 1974.

0074 PARTICIPATION OF HORMONAL FACTORS IN THE REGULATION OF MOTOR ACTIVITY IN THE DUODENUM. *(Rus.)* Fokina, A. A.; Slobozhankin, A. D.; Solovieva, I. A. (I. P. Pavlov Inst. Physiol., Leningrad, USSR). *Fiziol. Zh. SSSR* 60(11):1703-1710, 1974.

0075 CHARACTERISTICS OF INTESTINAL MOTOR ACTIVITY FROM BALLONOGRAPHIC FINDINGS AND RECORDINGS OF LUMINAL PRESSURE WITH AN OPEN CATHETER. *(Rus.)* Galperin, Iu. M.; Popova, T. S. (N. V. Sklifosovskii Sci. Res. Inst. Emerg. Med. Treat., Moscow, USSR). *Fiziol. Zh. SSSR* 60(10):1582-1591, 1974.

0076 IN VITRO STUDY OF THE ELECTROMYOGRAPHIC ACTIVITY OF THE PROXIMAL AND DISTAL COLON IN THE RABBIT. *(Fr.)* Jule, Y. (Inst. Neurophysiol. Psychophysiol., Marseille, France). *J. Physiol. (Paris)* 68(3):305-329, 1974.

0077 EFFECT OF 6-HYDROXYDOPAMINE ON [^{3}H]-ADENINE NUCLEOTIDE AND [^{3}H]-NORADRENALINE RELEASE FROM THE GUINEA-PIG TAENIA CAECUM EVOKED BY ELECTRICAL FIELD STIMULATION, NICOTINE AND PERIVASCULAR NERVE STIMULATION. *(E.)* Kuchii, M.; Miyahara, J. T.; Shibata, S. (Sch. Med., Univ. Hawaii, Honolulu). *Br. J. Pharmacol.* 51(4):577-583, 1974.

0078 13-NORLEUCINE AND MOTILIN: ANALYSIS OF THE EFFECTS ON THE GASTROINTESTINAL TRACT. *(Ger.)* Domschke, W.; Strunz, U.; Mitznegg, P.; Ruppin, H.; Domschke, S.; Schubert, E.; Wunsch, E.; Jaeger, E.; Demling, L. (Med. Clin. Polyclin., Univ. Erlangen, Germany). *Naturwissenschaften* 61(8):370, 1974.

0079 ACTIONS OF PUTATIVE TRANSMITTERS IN THE CHICKEN VAGUS NERVE/OESOPHAGUS AND REMAK NERVE/RECTUM PREPARATIONS. *(E.)* Bartlet, A. L. (Dep. Vet. Pharmacol., Edinburgh, Scotland). *Br. J. Pharmacol.* 51(4):549-558, 1974.

See also, 0387, 0431.

SECRETION AND METABOLISM

0080 DEVELOPMENT OF THE RAT SALIVARY GLANDS. IV. AMYLASE AND RIBONUCLEASE ACTIVITY DURING EMBRYONIC AND NEONATAL DEVELOPMENT OF THE PAROTID AND SUBMAXILLARY GLANDS. *(E.)* Ball, W. D. (Dep. Zool., Univ. Washington, Seattle). *Dev. Biol.* 41(2):267-277, 1974.

0081 CIRCADIAN RHYTHMS IN THE FLOW RATE AND COMPOSITION OF UNSTIMULATED AND STIMULATED HUMAN SUBMANDIBULAR SALIVA. *(E.)* Dawes, C. (Fac. Med., Univ. Manitoba, Winnipeg, Canada). *J. Physiol. (Lond.)* 244(2):535-548, 1975.

0082 SECRETION GRANULES OF THE RABBIT PAROTID GLAND: ISOLATION, SUBFRACTIONATION, AND CHARACTERIZATION OF THE MEMBRANE AND CONTENT SUBFRACTIONS. *(E.)* Castle, J. D.; Jamieson, J. D.; Palade, G. E. (Rockefeller Univ., New York, N.Y.). *J. Cell. Biol.* 64(1):182-210, 1974.

0083 ISOLATION OF A DEOXYRIBONUCLEASE FROM BOVINE PAROTID GLAND. *(E.)* Mizutani, T.; Mizutani, A. (Fac. Pharm. Sci., Nagoya City Univ., Japan). *Chem. Pharm. Bull. (Tokyo)* 22(11):2723-2725, 1974.

0084 IMMUNOCHEMICAL ASSAY OF SALIVA PAROTIN A. *(E.)* Shinoda, M.; Takagi, Y. (Hoshi Coll. Pharm., Tokyo, Japan). *Chem. Pharm. Bull. (Tokyo)* 22(11):2732-2734, 1974.

0085 AN EFFECT OF PROSTAGLANDIN E_1 ON THE ACINAR CELL OF THE RAT PAROTID GLAND. *(E.)* Lillie, J. H. (Med. Sch., Univ. Michigan, Ann Arbor). *J. Ultrastruct. Res.* 49(1):50-59, 1974.

0086 CHANGES IN NEURONAL UPTAKE AND METABOLISM OF, AND SENSITIVITY TO, NOREPINEPHRINE DURING THE DEGENERATION SECRETION IN THE RAT SUBMAXILLARY GLAND. *(E.)* Stefano, F. J. E.; Perec, C. J.; Tumilasci. O. R. (Natl. Counc. Sci. Tech. Invest., Buenos Aires, Argentina). *J. Pharmacol. Exp. Ther.* 191(3):403-417, 1974.

0087 RECURRENT DEGRADATION BY Canavalia ensiformis GLYCOSIDASES OF A POLYSACCHARIDE MOIETY FROM A GLYCOPROTEIN ISOLATED FROM HUMAN PAROTID SALIVA. *(Fr.)* Boersma, A.; Lamblin, G.; Degand, P. (Res. Unit Protein Biochem., INSERM, Lille, France). *C. R. Acad. Sci. [D] (Paris)* 279(12):1031-1034, 1974.

0088 IN VIVO DRUG METABOLISM AFTER CHRONIC ETHANOL ADMINISTRATION: INFLUENCE OF DIETARY FAT. *(E.)* Hetu, C.; Joly, J. G. (St. Luke Hosp., Montreal, Canada). *Gastroenterology* 67(4):798, 1974.

0089 EFFECT OF TWO VITAMIN D_3 METABOLITES, DIHYDROXY-24,25-CHOLECALCIFEROL AND DIHYDROXY-25,26-CHOLECALCIFEROL, ON THE MOST IMPORTANT PARAMETERS OF CALCIUM METABOLISM IN THE RAT. *(Fr.)* Milhaud, G.; Labat, M. L.; Redel, J. (Fac. Med., Paris, France). *C. R. Acad. Sci. [D] (Paris)* 279(10):827-830, 1974.

SECRETION AND METABOLISM

Stomach

0090 THE RELEASE OF HISTAMINE DURING GASTRIC ACID SECRETION IN CONSCIOUS RATS. *(E.)* El Munshid, H. A.; Lake, H. J. (Univ. Med. Sch., Newcastle upon Tyne, England). *J. Physiol. (Lond.)* 242(3):571-587, 1974.

Various groups of conscious gastric-cannulated adult female Wistar rats were given ^{3}H-histidine and aminoguanidine by dosage procedures intended to build up fast-turnover and slow-turnover pools of tissue ^{3}H-histamine to study histamine release during gastric acid secretion. Slow-pool turnover conditions were established by giving daily s.c. injections of ^{3}H-histidine (4 µC) and aminoguanidine (10 mg/kg) to 10 rats sacrificed at intervals over a period of 18 days, 24 hr after the final injection. The fast-turnover pool was established by giving continuous i.v. infusions of ^{3}H-histidine (4 µC/hr) and aminoguanidine (1 mg/kg/hr) to 10 rats fasted overnight. The animals were sacrificed in pairs at intervals over a 20-hr period. The amount of ^{3}H-histamine in gastric juice derived from either the slow- or fast-turnover pools was very low and was not altered during stimulation of gastric acid secretion by 3.3 µg/kg/min i.v. pentagastrin. Pentagastrin-induced urinary excretion of labeled histamine from the slow-turnover pool was abolished by gastrectomy (5 rats), and the ^{3}H-histamine liberated by pentagastrin from this pool appeared to have been derived from the stomach. Evidence was not found for the existence of a slow-turnover histamine pool in the glandular mucosa of the stomach; thus, the source of the pentagastrin-liberated histamine within the stomach was uncertain. Pentagastrin (3.3 µg/kg/min i.v.) did not cause an increased urinary excretion of ^{3}H-histamine from the fast-turnover pool. The amount of ^{3}H-histamine excreted by gastrectomized rats was not different from that produced by gastric-cannulated animals, suggesting that a high proportion of urinary histamine derived from the

fast-turnover pool was non-gastric in origin. Although the urinary output of ^{3}H-histamine was prolonged beyond the end of pentagastrin administration and gastric acid secretion in some experiments, the overall data did not suggest that urinary histamine output and gastric acid secretion take different time courses.

0091 THE ROLE OF HISTAMINE RECEPTORS IN THE STIMULATION OF GASTRIC ACID SECRETION. *(E.)* Konturek, S. J.; Demitrescu, T.; Radecki, T. (Inst. Physiol., Med. Acad., Krakow, Poland). *Am. J. Dig. Dis.* 19(11):999-1006, 1974.

In 7 conscious cats provided with chronic gastric fistulas 6 months previously, metiamide, an H_2-receptor antagonist, when given in a dose of 12 μM/kg/hr, inhibited gastric acid output stimulated maximally with histamine by 70%, pentagastrin by 55%, cerulein by 75%, insulin by 67%, urecholine by 100% and peptone meal by 95%. Atropine sulfate administered in a dose of 0.288 μM/kg/hr also inhibited gastric response to all these stimulants except histamine. These results can be explained in 3 ways: 1) all secretagogues act *via* histamine, and metiamide blocks the action of this final common chemostimulator; 2) the receptors for histamine, gastrin, and acetylcholine interact on the parietal cells and the inhibition of histamine receptors by metiamide decreases the affinity of the other 2 receptors to their antagonists; or 3) metiamide is a nonselective inhibitor of parietal cell receptors.

0092 EFFECTS OF PROPRANOLOL ON GASTRIC SECRETION IN ALBINO RATS. *(E.)* Debnath, P. K.; Gode, K. D.; Das, D. G.; Sanyal, A. K. (Inst. Med. Sci., Banaras Hindu Univ., Varanasi, India). *Br. J. Pharmacol.* 51(2):213-216, 1974.

The effects of graded doses of propranolol (0.5-50.00 mg/kg i.p.) on gastric secretion and pyloric-ligated ulcers were studied in inbred albino rats of both sexes. In all experiments, propranolol was administered 30 min before pyloric ligation for 4 hr. A dose of 1 mg/kg propranolol caused a significant increase and 50 mg/kg caused a significant inhibition of the volume (ml/100 g), total acid output (μM/4 hr), and total peptic activity (μM tyrosine/4 hr) of the gastric juice. Although total acid output decreased at the highest dose of propranolol, there was no significant change in peptic activity/ml gastric juice. The effect of propranolol on pyloric-ligated ulcers was also dose dependent: 1 mg/kg caused an increase while 50 mg/kg produced a significant decrease in the incidence of pyloric-ligated ulcers. This finding suggests that the ulcerogenic mechanism in pyloric-ligated rats is directly proportional to the total acid and pepsin output.

0093 PURE HUMAN MINIGASTRIN: SECRETORY POTENCY AND DISAPPEARANCE RATE. *(E.)* Debas, H.; Walsh, J. H.; Grossman, M. I. (VA Wadsworth Hosp. Cent., Los Angeles, Calif.). *Gut* 15(9):686-689, 1974.

Pure human natural nonsulfated minigastrin (HG-13-I) and pure human natural nonsulfated heptadecapeptide gastrin (HG-17-I) were compared with regard to acid-stimulating potency and rate of disappearance from the blood in 3 dogs with gastric fistulas and Heidenhain pouches given continuous i.v. infusion in doses of 100, 200, 400, and 800 pM/kg/hr. Equimolar infusion rates of HG-13-I and HG-17-I caused equimolar increases over basal of serum immunoreactive gastrin, though HG-13-I was less than half as potent as HG-17-I in stimulating acid secretion (potency ratio 0.4; 95% confidence limits 0.2-0.6). Clearance rates, calculated by dividing the dose infused by the plateau blood level of gastrin, were similar for the 2 gastrins (79 ml/kg/min mean value for HG-13-I and 83 ml/kg/min for HG-17-I). The half time for disappearance of HG-13-I from the blood was 1.75 min and its volume of distribution was calculated to be 0.17 liter/kg, values similar to those found for HG-17-I in an earlier study (2.9 min and 0.24 liter/kg, resp.). It is suggested that these differences may not be significant, but the problem requires further study.

0094 EFFECT OF GANGLIONIC AND MUSCARINIC BLOCKADE ON MECHANISM OF PEPSIN-SECRETION STIMULATION. *(E.)* Magee, D. F.; Dutt, B. (Creighton Univ. Sch. Med., Omaha, Nebr.). *Am. J. Physiol.* 227(5):1178-1180, 1974.

The effect of ganglionic and muscarinic blockade on the stimulation of pepsin secretion was examined in conscious Labrador dogs with gastric fistulas and Heidenhain pouches. Gastrin pentapeptide (GP) in doses up to 2 μg/min i.v. did not stimulate pepsin secretion from denervated fundic pouches (5 dogs), while atropine (1 mg i.v.) and pentolinium tartrate (1 mg/kg i.m.) reduced basal pepsin output by about 50%. GP (1 μg/min) was as effective on acid secretion after the ganglionic blocking agent (pentolinium tartrate) as without it, whereas atropine depressed GP-stimulated pouch acid from about 200-20 mEq acid. The usual GP-stimulation of fistula pepsin secretion (5 dogs) was abolished by both pentolinium and atropine, though acid secretion was unchanged by pentolinium and reduced by atropine from about 250 to 50 mEq. Atropine (0.5 mg; 5 dogs) virtually abolished pilocarpine (40 μg/min) stimulation of acid and pepsin in the denervated pouch, while pentolinium had no effect on either pepsin or acid. Atropine (0.5 mg; 5 dogs) abolished the acid and pepsin response to pilocarpine (40 μg/min) for gastric fistula, while ganglionic blockade (1 mg/kg pentolinium) greatly reduced, but did not abolish, the dose-related pilocarpine stimulation of pepsin (from 400 to 150 pepsin U/10 min collection) and acid (800 to 250 mEq). The acid data were not exact since contamination with saliva was great. Secretin (up to 2 secretin U/min) did not stimulate acid secretion from either pouch or fistula (6 dogs) but was a potent pepsin stimulant in both, raising the pepsin output by about 100 U in each case. Pentolinium (5 mg) raised secretion of pouch pepsin from 63.8 to 96.6 U, while 0.25 mg atropine i.v. reduced pepsin secretion from 76.6 to 58.3 U. Secretin stimulation (up to 2 secretin U/min; 6 dogs) of fistula as a rule resulted in re-

gurgitation of duodenal juice into the stomach. Ganglionic blockade (1 mg/kg i.m. pentolinium) did not alter the secretin-pepsin dose-response curve for fistula, while atropine (1 mg i.v.) abolished the secretin-pepsin dose-response relationship. Both secretin and pilocarpine appear to act directly on the chief cell *via* a muscarinic mechanism, whereas GP appears to act indirectly, possibly *via* a nicotinic ganglionic mechanism that is absent in the Heidenhain pouch. In contrast, GP and pilocarpine, as determined by muscarinic blockade, appear to act similarly in both pouch and fistula; secretin does not stimulate acid in either pouch or fistula.

0095 GAS CHROMATOGRAPHIC-MASS SPECTROMETRIC DETERMINATION OF N-NITROSODIMETHYLAMINE FORMED IN SYNTHETIC AND HUMAN GASTRIC JUICE. *(E.)* Lane, R. P.; Rice, R. H.; Bailey, M. E. (Dep. Food Sci. Nutr., Univ. Missouri, Columbia). *J. Agric. Food Chem.* 22(6):1019-1023, 1974.

An analytical procedure for the determination of N-nitrosodimethylamine in human gastric juice is described. The method involves alkalization of gastric juice with potassium hydroxide, reflux for 4 hr followed by acidification with sulfuric acid/sodium sulfate and extraction with chloroform. The chloroform extract is reextracted with sodium hydroxide and the solution is concentrated by evaporation. The extracts are eluted on an acidic Celete column with chloroform. N-nitrosodiethylamine is added as an internal standard and N-nitrosodiethylamine levels are determined by gas chromatography and confirmed by low and high-resolution mass spectroscopy. The method allows detection of nitrosamine concentrations as low as 0.1 ppb. The recovery of 2.5-10.0 µg N-nitrosodiethylamine from 10, 100 ml aqueous solution samples was 76-96% (mean 87%) and that from 3, 100 ml human gastric juice samples containing 5 µg averaged 79% (76-82%). Glycine (1.3 mM) inhibited nitrosamine formation in gastric juice by about 80% in the presence of 5 µg and by 87% in the presence of 10 µg diethylamine at pH 3.4. Optimal pH for nitrosamine formation in human gastric juice was 2.5 instead of 3.4 as predicted from nitrous anhydride reactions. An amino acid mixture inhibited nitrosamine formation at pH 2.5 and stimulated formation at pH 4.5. Aqueous meat extract inhibited nitrosamine formation by about 30% at pH 2.5 and 4.5. It should thus be possible to detect and confirm whether or not N-nitrosodimethylamine is formed *in vivo* after digestion of cured meats or other food materials.

0096 GASTRIC MUCOSAL ENERGY METABOLISM AND "STRESS ULCERATION". *(E.)* Menguy, G.; Masters, Y. F. (Univ. Rochester Sch. Med., N.Y.). *Ann. Surg.* 180(4):538-548, 1974.

Gastric mucosal histologic and energy metabolism changes were studied in rats and rabbits in which hemorrhagic shock was induced by acute blood loss. In rats in which hemorrhagic shock was induced by removing 20 ml/kg blood from the vena cava in 2 min, focal, superficial epithelial gastric necrosis was present after 15 min, and by 60 min all animals had focal mucosal hemorrhage with superficial erosions ("stress ulcers") in the corpus and fundus but not in the antrum. Associated with these changes was a 75% decrease in gastric mucosal ATP and a 47% drop in total mucosal adenosine phosphates. Production of warm ischemia for 30-60 sec in the stomachs of rats by complete tissue severance resulted in a 50% drop in gastric mucosal adenosine phosphates within 15 sec. In addition, gastric mucosal glycogen was decreased by 60% after 60 sec of ischemia. Comparative studies of the antrum, corpus and fundus mucosas of rabbits acutely bled to 30 mm Hg blood pressure showed a more rapid breakdown of high energy phosphate as well as a more rapid glycolytic flux in the corpus and fundus than in the antrum. Dephosphorylation of adenosine phosphates in the ischemic corpus and fundus mucosa of rabbits in shock was 2-fold greater than in the ischemic antrum. Similar studies using fasted rabbits showed that a greater mucosal energy deficit was accompanied by more severe gastric mucosal injury. When the bile salt, sodium taurocholate (30 mM) was present in the stomach of shocked rabbits, gastric mucosal ulceration was more severe, the mucosal energy deficit was greater, mitochondrial oxidative phosphorylation was uncoupled and $(Na^+ + K^+)$-ATPase activity of corpus mucosal homogenates was completely inhibited. These studies are consistent with the hypothesis that an energy deficit severe enough to cause cellular necrosis is the event linking shock-induced gastric mucosal ischemia and stress ulceration.

0097 PROLIFERATION OF THE GLANDULAR EPITHELIUM IN THE RESECTED STOMACH OF RATS. *(Rus.)* Timashkevich, T. B.; Zaporozhchenko, I. G. (Inst. Hum. Morphol., Moscow, USSR). *Biull. Eksp. Biol. Med.* 78(7):94-98, 1974.

Forty 200-300 g male rats had 45-50% of the fundal wall resected. Pulse labeling and thymidine-^{3}H injection every 5 hr within 24 hr (0.3 µCi/kg) indicated an increase in DNA synthesizing and dividing cells of all types in the chief peptic glands within the first 3 days after the resection. In 5-10 days cell proliferation was slower than in the intact animals, except DNA synthesizing parietal cells. After 3-6 months the daily number of DNA-synthesizing cells of all types reached the level of the intact animals. There was an increase in the surface and crypt epithelium cells, and mucous neck and parietal cells. Partial reduction and regression to a more primitive level of differentiation was observed in peptic cells.

0098 FUNCTIONAL AND MORPHOLOGICAL RECONSTRUCTION OF THE GASTRIC GLANDULAR APPARATUS IN DISORDERS OF THE EXOCRINE PANCREAS AND EXPERIMENTAL REFLUX PANCREATITIS. *(Rus.)* Blagovidov, D. F.; Pomel'tsov, A. N.; Shatalov, V. N.; Gerasimova, O. B. (4th Princ. Off., Minist. Health USSR, Moscow). *Biull. Eksp. Biol. Med.* 78(7):33-36, 1974.

Studies were made of the fundal mucosa in dogs with excluded exocrine pancreas and experimental reflux pancreatitis. Findings indicate a substantial increase of the chief cells and decrease of the parietal cells in the fundal gastric glands within 6-17 months after suturing the pancreatic ducts. Elevated

RNA concentration was observed in the chief cells. The morphological changes in the peptic glands were stable throughout the 20-month experiment, and fully correlated with the previous data on the changes in the gastric function brought on by the disruption of the exocrine pancreas: higher peptic capacity of the gastric juice was obviously related to the numerical increase of the chief cells in the fundal mucosa, while the acidity drop was the result of the fewer parietal cells. The functional and morphological readjustment of the peptic glands in experimental reflux pancreatitis and occlusion of the pancreatic ducts may be a compensatory reaction to the disruption of the exocrine pancreatic function.

0099 THE CONTAMINATION OF GASTRIC JUICE BY THE MUCOUS SECRETIONS OF THE UPPER DIGESTIVE TRACT. *(E.)* Lambert, R. (INSERM, Edward Herriot Hosp., Lyon, France). *Rend. Gastroenterol.* 6(1):60-64, 1974.

0100 ACTION MECHANISMS OF SUBSTANCES INHIBITING GASTRIC ACID SECRETION. *(Fr.)* Bali, J. P.; Pham-Thanh-Chi (Fac. Pharm., Montpellier France). *Trav. Soc. Pharm. Montpellier* 34(3):197-206, 1974.

0101 EFFECT OF INCREASING DOSES OF PENTAGASTRIN ON ACID SECRETION IN PERFUSION EXPERIMENTS ON THE RAT STOMACH FOLLOWING VAGOTOMY. *(Ger.)* Struve, C. W.; Hantschmann, N. (Surg. Clin., Univ. Kiel, Germany). *Res. Exp. Med. (Berl.)* 163(4):289-298, 1974.

0102 INFLUENCE OF SPLENECTOMY ON GASTRIC JUICE SECRETION AND ULCERATION IN RATS. *(Ger.)* Koch, G.; Schumpelick, V.; von Rehren, D. (Surg. Clin., Univ. Hamburg, Germany). *Langenbecks Arch. Chir.* 336(1):15-23, 1974.

0103 SERUM GASTRIN IN UREMIC PATIENTS DURING HEMODIALYSIS. *(Ger.)* Pointner, H.; Flegel, U.; Schmidt, P.; Zazgornik, J.; Kopsa, H.; Waldhausl, W. (1st Med. Clin., Univ. Vienna, Austria). *Wien. Klin. Wochenschr.* 86(22):696-699, 1974.

0104 SERUM GASTRIN AFTER A TEST MEAL IN PATIENTS UNDERGOING RENAL DIALYSIS. *(Ger.)* Pointner, H.; Flegel, U.; Schmidt, P.; Zazgornik, J.; Kopsa, H.; Waldhausl, W. (1st Med. Clin., Univ. Vienna, Austria). *Wien. Klin. Wochenschr.* 86(22): 699-702, 1974.

See also, 0018, 0448, 0730.

SECRETION AND METABOLISM

Pancreas

0105 INDUCTION OF CHYMOTRYPSINOGEN BY HYDROCORTISONE IN EMBRYONIC CHICK PANCREAS IN VITRO. *(E.)* Cohen, A.; Kulka, R. G. (Dep. Biol. Chem., Heb. Univ. Jerusalem, Israel). *J. Biol. Chem.* 249(14):4522-4527, 1974.

Stimulation of chymotrypsinogen accumulation by hydrocortisone was studied *in vitro* in pancreas explants from 9-day-old chick embryos. After incubation with hydrocortisone (10^{-6} M for 22 hr), 75% of the chymotrypsinogen was released into the medium. After 45 hr of incubation, 73% of the enzyme was released. A lag of 10-15 hr in response of pancreas explants to hydrocortisone was noted. The dose-response curve for chymotrypsinogen showed that the latter increased with hydrocortisone concentration and reached a maximum when the hormone concentration was about 8 x 10^{-7} M. The threshold of hormone concentration was 2-4 x 10^{-8} M. Radioimmunoprecipitation studies showed that the hormone selectively stimulated *de novo* synthesis of chymotrypsinogen. The rate of synthesis increased with the time of incubation. The net chymotrypsinogen cpm/total cpm x 100 was 0.92 after 15 hr, 2.33 after 24 hr, and 3.19 after 48 hr. Exposure of pancreas explants to hydrocortisone for 3 hr followed by washing out of the hormone and incubation for 48 hr gave the same degree of incubation of the enzyme (23.6 U/pancreas x 10^3) as continuous exposure to the hormone for 48 hr (27.9 U/pancreas x 10^3). Actinomycin D (50 µg/ml) was added at zero time and hydrocortisone (10^{-6} M) was added 1/2 hr later. Actinomycin D inhibited the induction of enzyme by 90-100% in the 9-day-old pancreas (18 hr incubation). The 12-day-old pancreas (20 hr incubation) showed an inhibition from 50-80%. A marked inhibition of enzyme synthesis was observed when actinomycin D was added at zero time or up to 5 hr after the addition of hydrocortisone. There was almost no effect on chymotrypsinogen accumulation when actinomycin D was added 10 hr after hydrocortisone.

0106 STUDY BY AUTORADIOGRAPHY OF THE TRANSPORT AND EXCRETION OF PANCREATIC ENZYMES IN THE ACINAR CELL OF ALCOHOLIC RATS. *(Fr.)* Lechene, P.; Sarles, H. (Res. Unit Dig. Pathol., INSERM, Marseille, France). *Biol. Gastroenterol. (Paris)* 7(1): 61-64, 1974.

The effect of alcohol on intracellular transport and excretion of enzymes in rat pancreatic acinar cells was studied by measuring the organelle content of labeled pancreatic proteins through electron microscopic autoradiography. For 4 months, Wistar rats were given 20% alcohol (4 animals) or water (4 animals) in association with a balanced diet. The rats

were sacrificed after a 24 hr fast, and pancreatic slices were labeled by 3-min incubation with ^{3}H-L-leucine 4,5, followed by incubation in a chased medium for 7, 17, 37, and 57 min. The graphs of autoradiographically measured enzymes *versus* time demonstrated no significant difference between alcoholic and control rats in the endoplasmic reticulum, Golgi bodies, and condensation vacuoles. For zymogen granules, the curves were essentially the same until 40 min when enzymes continued to increase in controls but decreased steadily in alcoholic rats (corresponding to measured increase in enzyme excreted). Thus in alcoholic rats there was increased enzyme excretion without any effect on intracellular transport.

0107 IN VIVO EFFECTS OF PANCREOZYMIN, SECRETIN, VASOACTIVE INTESTINAL POLYPEPTIDE, AND PILOCARPINE ON THE LEVELS OF CYCLIC AMP AND CYCLIC GMP IN THE RAT PANCREAS. *(E.)* Robberecht, P.; Deschodt-Lanchman, M.; De Neef, P.; Borgeat, P.; Christophe, J. (Brussels Univ. Sch. Med., Belgium). *FEBS Lett.* 43(2):139-143, 1974.

Pancreatic secretion of amylase in response to pancreozymin and pilocarpine and the time course of cyclic nucleotide levels was investigated *in vivo* in male Wistar rats. Cyclic AMP was estimated by protein binding assay and cyclic GMP was determined by radioimmunoassay. Pancreozymin (0.33 µg in 0.2 ml 0.9% NaCl, i.v.) caused a rapid increase in amylase secretion which remained high at 30 min following injection and declined to control values after 60 min. The same amount of pancreozymin caused no change in the levels of cyclic AMP (cAMP) during the 15 min following injection. Pilocarpine (30 mg/kg, s.c.) caused no apparent change in levels of cAMP. However, animals injected i.v. with secretin (2.75 µg/kg) or vasoactive intestinal polypeptide (VIP) (2.75 µg/kg in 0.2 ml 0.9% NaCl), caused the cAMP level to double. Cyclic AMP increased in 30 to 60 sec and declined to control value in 10-15 min. Pilocarpine (30 mg/kg, s.c.) and pancreozymin (0.33 µg in 0.2 ml 0.9% NaCl, i.v.) caused a 2-fold increase in cyclic GMP levels at 5-6 min after injection, and decreased sharply thereafter, declining to control levels after about 10-15 min. These results support the role of cAMP as second messenger of secretin, and they suggest also that the hormonal receptor sites of secretin and VIP are connected to the same adenylate cyclase. The rise of cyclic GMP may be due to an increase in protein phosphorylation which is associated with the stimulation of amylase.

0108 INHIBITION OF PANCREATIC EXOCRINE SECRETION BY INTRA-COLONIC OLEIC ACID INFUSION IN THE DOG. *(E.)* Hage, G.; Tiscornia, O.; Palasciano, G.; Sarles, H. (Dig. Pathol. Unit, INSERM, Marseille, France). *Biomedecine* 21(6):263-267, 1974.

The effects of an oleic acid perfusion in the proximal colon on exocrine pancreatic secretion was studied in 3 dogs, surgically prepared with gastric, duodenal, ileal, and colonic fistulas. Secretin (1.0 U/kg/hr, i.v.) was infused by catheter, and maintained at a constant rate of 100 ml/hr. When the flow rate of pancreatic secretion became constant, oleic acid (20 ml) was perfused for 10 min into the proximal colon and changes in pancreatic secretion were evaluated for 80 to 100 min. In a control series, oleic acid (20 ml) was injected into the distal ileum. Volume, bicarbonate concentration, bicarbonate output, protein concentration and protein output were measured every 20 min. Gastric levels were determined before and in every 20 min sample following the ileal or colonic oleic acid perfusion. There was a marked and prompt decrease of pancreatic secretion after oleic acid infusion into the colon. Volume decreased 40%; bicarbonate output, 47%; and protein output, 56%. Protein concentration did not decrease significantly. Gastrin and fatty acid blood levels did not change, and saline did not affect the pancreatic secretion or gastrin level. There was an increase in pancreatic secretion for the first 60 min following infusion of oleic acid into the ileum. Protein output increased 80% and remained significant for 40 min. Volume increased 30%, but bicarbonate output only increased 28%. Gastrin levels did not increase after infusion and instilled saline did not affect any of the parameters studied. These results demonstrate that oleic acid infusion into the colon has an inhibitory effect on exocrine pancreas secretion. It is suggested that pancreatic secretion decreases during digestion, due to the arrival of digestion products into the colon.

0109 FUNCTION OF THE EXOCRINE PANCREAS IN THE RAT: INHIBITION BY LARGE DOSES OF CHOLECYSTOKININ-PANCREOZYMIN AND ANESTHESIA. *(Ger.)* Hotz, J.; Neumann, K.; Zoller, A.; Ziegler, R.; Goebell, H. (Dep. Intern. Med., Univ. Ulm, Germany). *Verh. Dtsch. Ges. Inn. Med.* 79:862-865, 1973.

The effect of large doses of cholecystokinin-pancreozymin on the trypsin secretion by the pancreas was studied in conscious and anesthetized male Wistar rats. Increasing doses of cholecystokinin-pancreozymin (1, 2, 4 and 8 U/100 g/10 min in i.v. infusion) caused progressive reduction of the trypsin secretion maxima and shortened duration of these maxima from 50 to 30 min during stimulation for 3 hr in rats anesthetized by i.m. administration of 300 mg urethane per 100 g. The absence of secreted granules in the acinus cell at the end of the stimulation is indicative of the progressive inhibition not only of the secretion but also of the production of trypsin. This paradoxical inhibition of the enzyme secretion by the same doses of cholecystokinin-pancreozymin was absent in conscious animals. Both urethane and pentobarbital inhibited the basal and stimulated pancreas secretion.

0110 CELLULAR CHANGES IN THE EXOCRINE PANCREAS OF RATS FED TWO LEVELS OF MAGNESIUM AND PROTEIN. *(E.)* Greger, J. L.; Schwartz, R. (Coll. Human Ecol., Cornell Univ., Ithaca, N.Y.). *J. Nutr.* 104(12):1610-1617, 1974.

The acute and chronic effects of magnesium deficiency on the pancreas were studied in male Sprague-Dawley rats fed a diet containing either 15 or 30% casein

and 50 ppm Mg^{++} for 60 days. Controls received a 15 or 30% casein diet containing 100 ppm Mg^{++}. Mg^{++} deficiency as determined by atomic absorption spectrophotometry occurred earlier in rats fed the 30% casein diet (day 15) than in those fed the 15% diet (day 35). Plasma Mg^{++} levels were significantly reduced by day 5 regardless of protein content of the diet. Plasma Ca^{++} showed no change. Blood histamine levels increased earlier and reached higher peak levels in Mg^{++} depleted rats fed the 30% casein diet. Pancreatic weights increased with time but showed no relation to the type of diet. Pancreatic Mg^{++} concentrations were also unaffected by diet. Pancreatic weight, RNA, protein and chymotrypsin levels were studied in 24-hr fasted and refed rats after 10 days of dieting. Pancreatic weights were highest in rats fed the 30% casein 100 ppm Mg^{++} diet. No differences occurred in fasted rats. No changes in RNA content were seen in any group. Protein content was greater in refed than fasted rats fed the 50 ppm Mg^{++} diet. Chymotrypsin levels were unaffected by dietary Mg^{++}. Incorporation of i.v. injected ^{14}C-valine into pancreatic protein was significantly greater after 2 hr in fasted rats fed the 100 ppm Mg^{++} than in those fed the 50 ppm Mg^{++} diet regardless of casein content. No such differences were observed in refed rats, and the differences in the fasted animals were at least in part due to differences in the rate of ^{14}C-valine transport into the pancreatic cells. The results of these studies confirm previous studies which also showed no change in pancreatic Mg^{++} content and function in Mg^{++} deficient animals.

0111 COMPARISON OF THE ACTIONS OF PORCINE SECRETION AND EXTRACTS OF CHICKEN DUODENUM ON PANCREATIC EXOCRINE SECRETION IN THE CAT AND TURKEY. *(E.)* Dockray, G. J. (Dep. Physiol., Univ. Liverpool, England). *J. Physiol. (Lond.)* 244(3):625-637, 1975.

0112 STIMULATION OF PANCREATIC ELECTROLYTE SECRETION BY CHOLERA TOXIN. *(E.)* Case, R. M.; Smith, P. A. (Univ. Med. Sch., Newcastle upon Tyne, England). *J. Physiol. (Lond.)* 244(1):28P-29P, 1975.

0113 THE ACTIVE SITE OF PANCREATIC PORCINE ELASTASE: SPECIFICITY, SIZE AND STEREOSPECIFICITY. *(E.)* Atlas, D. (Weizmann Inst. Sci., Rehovot, Israel). *Isr. J. Chem.* 12(1/2):455-469, 1974.

0114 EFFECT OF SYNTHETIC PROTEASE INHIBITORS ON THE ACTION AND ACTIVATION OF PROTEOLYTIC PANCREATIC ENZYMES. *(Ger.)* Markwardt, F.; Walsmann, P.; Hoffmann, J. (Med. Acad., Erfurt, Germany). *Acta Biol. Med. Ger.* 32(5):433-439, 1974.

0115 PARTIAL PURIFICATION AND STUDY OF THREE PROTEIN KINASES FROM RAT PANCREAS. *(Fr.)* Cenatiempo, Y.; Marchis-Mouren, G. (Inst. Biol. Chem., Univ. Aix-Marseille, France). *C. R. Acad. Sci. [D] (Paris)* 279(20):1633-1636, 1974.

0116 LONG-TERM EXOCRINE FUNCTION OF THE PANCREAS TRANSPLANT. *(E.)* Charters, III, A. C.; Lee, S.; Storck, G.; Chandler, J. G.; Orloff, M. J. (Univ. California Sch. Med., San Diego). *Am. J. Surg.* 129(1):16-22, 1975.

0117 ENZYMATIC EFFECTIVENESS OF PANCREATIC SUBSTITUTION THERAPY BY DRUGS AND SUGAR-COATED PILLS. *(Ger.)* Malis, F.; Premysl, F. (2nd Gastroenterol. Res. Dep., Charles Univ., Prague, Czechoslovakia). *Z. Gesamte Inn. Med.* 29(14):569-576, 1974.

See also, 0098.

SECRETION AND METABOLISM

Hepatobiliary Tract

0118 EFFECT OF DIETARY CHENODEOXYCHOLIC ACID AND LITHOCHOLIC ACID IN THE RABBIT. *(E.)* Fischer, C. D.; Cooper, N. S.; Rothschild, M. A.; Mosbach, E. H. (Public Health Res. Inst., New York, N.Y.). *Am. J. Dig. Dis.* 19(10):877-886, 1974.

The feeding of chenodeoxycholic acid or lithocholic acid (0.05 or 0.5% of the diet) to rabbits produced cirrhotic and necrotic changes in the liver, accompanied by an increase of secondary bile acids in bile. Animals fed 0.05% lithocholic acid, 0.05% or 0.5% chenodeoxycholic acid, but not those receiving 0.5% lithocholic acid were able to survive for a period of 21 days. The most severe cirrhotic and necrotic changes were observed in the rabbits fed either 0.5% lithocholic acid or 0.5% chenodeoxycholic acid for 6 days or longer. These changes included focal hepatocyte necrosis, bile duct proliferation, lymphocytic, histiocytic and occasionally neutrophilic portal infiltrations and portal fibrosis. Liver damage appeared to correlate with bile composition but not with hepatic cholesterol content. The pathologic involvement was greatest whenever the percentage of lithocholic acid in the bile exceeded 15%. It is concluded that chenodeoxycholic acid (which is not a major constituent of rabbit bile) exerts its hepatotoxic effects largely because it is converted to lithocholic acid by the intestinal bacterial flora.

0119 ACTIVITIES OF PROLYL HYDROXYLASE, LYSYL HYDROXYLASE, COLLAGEN GALACTOSYLTRANSFERASE AND COLLAGEN GLUCOSYLTRANSFERASE IN THE LIVER OF RATS WITH HEPATIC INJURY. *(E.)* Risteli, J.; Kivirikko, K. I. (Dept. Med. Biochem., Univ. Oulu, Finland). *Biochem. J.* 144(1):115-122, 1974.

The activities of prolyl hydroxylase, lysyl hydroxylase, collagen galactosyltransferase and collagen glucosyltransferase, four enzymes catalyzing post-translational modifications of the collagen polypeptide chains, were assayed in the livers of rats with experimental hepatic injury. The liver injury was induced by injecting CCl_4 (0.05 ml/100 g) twice weekly, and assays of the enzymic activities were carried out in liver homogenates preincubated in Triton X-100 2 and 4 weeks after commencement of administration of CCl_4. The activities of all four enzymes had increased by 2 weeks after commencement of CCl_4 administration. This increase preceded an increase in the collagen content of the liver. A further slight increase was found in three of the enzyme activities during the subsequent 2 weeks of the experiment, whereas no further increase was found in the collagen galactosyltransferase activity. A statistically significant correlation was found between all four enzyme activities, but the magnitude of the increases varied considerably. The largest increase was found in lysyl hydroxylase activity, and at 4 weeks the magnitude of this was about 3 times that of the collagen galactosyltransferase activity. The results thus indicate that the increased enzyme activities cannot be explained simply by an increase in the number of collagen-producing cells having similar enzyme activity patterns to those of the cells initially present in the liver.

0120 RESTORATION OF HEPATIC MITOCHONDRIA DURING RECOVERY FROM CARBON TETRACHLORIDE INTOXICATION. *(E.)* Brabec, M. J.; Gray, R. H.; Bernstein, I. A. (Univ. Michigan, Ann Arbor). *Biochem. Pharmacol.* 23(23):3227-3238, 1974.

The functional and morphological recovery of rat hepatocyte mitochondria was studied following liver injury induced by a single i.p. injection of CCl_4 (1.4 ml/kg). Hepatocyte mitochondrial damage, which occurred beginning about 35 hr after CCl_4 injection, was characterized by a loss of respiratory activity, loss of phosphorylation coupled to respiration, and mitochondrial swelling attended by loss of cristae structure. Within 15-25 hr after full development of the mitochondrial lesion, the function and structure of the mitochondria were largely restored. Studies of the turnover of mitochondrial DNA and the rates of synthesis of mitochondrial DNA and protein indicated that the CCl_4-insulted hepatocyte is repairing the mitochondrial damage by the insertion of specific elements into the damaged organelle, rather than by proliferation of undamaged mitochondria for replacement. The failure of ethidium bromide, oxytetracycline and chloramphenicol, specific inhibitors of mitochondrial protein, and/or nucleic acid synthesis, to block this restoration substantiates the postulated repair process, and also indicates the noncritical nature of the respective mitochondrial functions during the repair process. Cytochrome measurements made during the period of acute damage revealed normal levels of cytochrome c, c_1 and aa_3. The observed elevation of cytochrome b is attributed to contamination of the preparation by hemoglobin.

0121 NATURE OF THE PROTECTION AGAINST CARBON TETRACHLORIDE-INDUCED HEPATOTOXICITY PRODUCED BY PRETREATMENT WITH DIBENAMINE [N-(2-CHLOROETHYL) DIBENZYLAMINE]. *(E.)* Maling, H. M.; Eichelbaum, F. M.; Saul, W.; Sipes, I. G.; Brown, E. A. B.; Gilette, J. R. (Natl. Heart Lung Inst., Bethesda, Md.). *Biochem. Pharmacol.* 23(10):1479-1491, 1974.

The plausible mechanisms by which Dibenamine [N-(2-chloroethyl) dibenzylamine] hydrochloride pretreatment (25 mg/kg s.c., 48 and 24 hr prior to administration of 1.0 mg/kg i.p. of CCl_4) protects rats against the hepatotoxic effects of CCl_4 have been elevated. Also, the distribution of ^{14}C-Dibenamine derivatives and their covalent binding to liver lipids and proteins (total and microsomal) has been studied at times of maximal protection against CCl_4-induced hepatotoxicity. Dibenamine pretreatment did not affect significantly the absorption of CCl_4 after i.p. administration or the elimination of CCl_4 from various tissues, as evident from unchanged tissue levels. Dibenamine pretreatment decreased the accumulation of the metabolite $CHCl_3$ in liver, the isopropanol-potentiated covalent binding of a small dose of $^{14}CCl_4$ (0.1 ml/kg) and the covalent binding of a necrosis-producing dose of $^{14}CCl_4$ (0.1 ml/kg) to proteins and lipids in liver, and the diene conjugation of liver microsomal lipids induced by CCl_4. These findings suggest that Dibenamine pretreatment impairs the microsomal enzymes which catalyze the formation of free radical derivatives of CCl_4. Dibenamine protection is not correlated with its concentration in liver or with the covalent binding of Dibenamine derivatives to liver microsomal protein.

0122 COLLAGENASE ACTIVITY IN EXPERIMENTAL HEPATIC FIBROSIS. *(E.)* Okazaki, I.; Maruyama, K. (Sch. Med., Keio Univ., Tokyo, Japan). *Nature* 252(5478):49:50, 1974.

The presence of collagenase in rat liver was demonstrated along with increased collagenase activity in experimental hepatic fibrosis induced by carbon tetrachloride. Fifty female Wistar rats received s.c. injections of 0.3 ml 50% CCl_4 in olive oil per 100 g body weight twice a week. Diced specimens of rat liver were incubated with collagen gels reconstituted from collagen that was salt extracted from the skin of young, growing Wistar rats. In 10 non-treated rats, 2 livers demonstrated weak collagenase activity, 5 livers minimal activity, and another 3 no appreciable lysis. In the CCl_4-treated rats, collagenolytic activity increased slightly in the initial stage of hepatic fibrosis (3 weeks after treatment) and markedly during extensive fibrosis (6 weeks). Collagenolytic activity in the irreversible cirrhotic stage (20 weeks) was poor compared with activity observed after 6 weeks. Under microscopic examina-

tion, livers from treated rats showed slight to moderate periportal fibrosis after 3 weeks, the formation of fibrous septa connecting portal tracts with the central canal after 6 weeks, and typical features of cirrhosis after 20 weeks. Collagenolytic activity was inhibited by the addition of sodium diaminetetraacetate (1×10^{-2} M final concentration). Neither aerobic nor anaerobic bacterial contamination was detected in gels where lysis was observed. Acrylamide gel electrophoresis of collagen incubated with pre-cirrhotic rat liver showed patterns similar to those seen with other animal collagenases. The increased collagenolytic activity was thus concluded to be due to collagenase. The proliferation of collagen fibers appeared to result from a more rapid synthesis of collagen in comparison to its resorption. Discontinuation of toxic stimuli presumably results in increased collagenolytic activity to resorb the excessive collagen fibers.

0123 HEMODYNAMIC EFFECTS OF CCl_4 IN THE INTACT LIVER OF THE CAT. *(E.)* Lautt, W. W.; Plaa, G. L. (Dep. Pharmacol., Univ. Montreal, Canada). *Can. J. Physiol. Pharmacol.* 52(3):727-735, 1974.

The hemodynamic changes in the liver of anesthetized cats was studied following CCl_4 administration. Blood flow in the intact liver did not change significantly over a period of 4 hr following intraduodenal injection of CCl_4 (1 ml/kg). Hepatocellular disruption, as determined by changes seen by the electron microscope, was well underway 2 hr after the injection. Twenty-four hours following an oral dose of CCl_4, the hepatic arterial resistance to blood flow was reduced and total blood flow to the liver was at least as high as in control animals. At this time, the hepatic artery appeared fully dilated and was less responsive to humoral (intra-arterial infusion of noradrenaline) and neural (reflex activation of the sympathetic nerves) constrictor influences. Thus, alterations in hepatic blood flow do not occur during the early phases of CCl_4-induced hepatic injury. These data indicate that diminished blood flow is not a causative factor in the initial phases of CCl_4-induced liver injury. By 24 hr, hepatic blood flow is altered in such a manner that the damaged liver receives a higher proportion of arterial blood and a total blood flow that is not reduced in spite of a generally depressed cardiovascular system.

0124 FAILURE OF PHENOBARBITAL TO INCREASE BILIRUBIN PRODUCTION IN THE RAT. *(E.)* Gisselbrecht, C.; Berk, P. D. (Natl. Inst. Arthritis Metab. Dig. Dis., Bethesda, Md.). *Biochem. Pharmacol.* 23 (20):2895-2905, 1974.

Bilrubin production, as measured by bile bilirubin excretion, in Sprague-Dawley rats after surgical creation of an external biliary fistula was assessed for groups of animals pretreated with phenobarbital (70 mg/kg i.p.) for 5 days prior to surgery, untreated rats, and rats receiving 4 days of phenobarbital treatment (70 mg/kg i.p.) 24 hr after surgery. Bile bilirubin excretion in 16 untreated animals averaged 0.71 mg/100 g/day during the 24-hr period following surgery. Despite the wide range (0.48-1.10 mg/100 g/day) in the group, values for each individual animal were highly consistent, averaging 101, 103, 93, and 92% of the initial 24-hr period baseline value during the 4 subsequent 24-hr collection periods. Bile bilrubin excretion in 15 rats pretreated with the drug for 5 days prior to surgery averaged 0.86 mg/100 g/day in the initial collection period. This did not represent a significant increase when compared to untreated controls. Values of bile bilirubin excretion likewise remained constant during subsequent collection periods during phenobarbital treatment. Although the initiation of phenobarbital therapy 24 hr after biliary surgery was associated with high mortality, adequate data were obtained for a group of 6 animals receiving 4 days of phenobarbital treatment (70 mg/kg i.p.). During the postoperative treatment period which was sufficient to produce a 2- to 3-fold increase in early labeling of bile bilrubin and a 3- to 4-fold increase in hepatic cytochrome P-450, bile bilirubin excretion averaged 105, 92, 80, and 100% of the baseline pretreatment value during 24-hr collection periods. Hence, phenobarbital-associated increases in early labeling of bile pigment and hepatic cytochrome P-450 content are not accompanied by an increase in total bilirubin excretion.

0125 INDUCTION BY PHENOBARBITAL OF HEPATIC MICROSOMAL DRUG-METABOLIZING ENZYME SYSTEM IN PARTIALLY HEPATECTOMIZED RATS. *(E.)* Hino, Y.; Imai, Y.; Sato, R. (Inst. Protein Res., Osaka Univ., Japan). *J. Biochem.* 76(4):735-744, 1974.

Phenobarbital induction (100 mg/kg, i.v.) of microsomal drug-metabolizing enzymes was studied in the residual portion of livers from partially hepatectomized male Sprague-Dawley rats. Phenobarbital injection 1 hr postoperatively resulted in a 150% rise in cytochrome P-450 over controls during the subsequent 48 hr. Sham-operated rats showed a 2-fold increase, comparable to phenobarbital-treated rats without partial hepatectomy. Preoperative capacity for phenobarbital-induced cytochrome P-450 increase was regained 4 days after partial hepatectomy, paralleling liver regeneration. Measurements of cytochrome P-450, NADPH-cytochrome c reductase, and cytochrome b_5, 48 hr after inducer injections given at various times postoperatively, showed decreases of 20-40% in the basal levels of the 3 enzymes within 5-7 days, though levels normalized to preoperative values within 2 weeks. Microsomal NADPH-cytochrome c reductase was not inducible by phenobarbital in either sham or partially hepatectomized rats immediately after surgery, though it gradually became inducible to a slight extent. Cytochrome b_5 remained essentially unaffected by phenobarbital administration at all liver regeneration stages and after sham operation in agreement with results obtained from controls. Changes in the ethyl isocyanide difference spectrum of reduced cytochrome P-450 following phenobarbital injection 4 days after partial hepatectomy were similar to those in inducer-treated sham-operated rats, although immediately after hepatectomy no changes were observed. The specific activity of aminopyrine N-demethylase of all microsomal prepara-

tions examined was linearly proportional to the specific content of cytochrome P-450.

0126 CHANGES IN IN VIVO METABOLISM OF BILE ACIDS IN RAT AFTER TREATMENT WITH PHENOBARBITAL. *(E.)* Cronholm, T.; Einarsson, K.; Gustafsson, J. A. (Dep. Chem., Karolinska Inst., Stockholm, Sweden). *Lipids* 9(11):844-849, 1974.

The influence of phenobarbital on pool size and turnover of bile acids was investigated in male Sprague-Dawley rats. Three rats were injected with phenobarbital (100 mg/kg body weight in 1 ml saline solution/day, i.p.); another group of 3 rats served as controls. After 10 days, all rats received i.p. injections of 0.1 mg [24-^{14}C]cholic acid and 0.5 mg tritium-labeled chenodeoxycholic acid. Phenobarbital-treated rats had a smaller cholic acid pool compared to control rats (6.08 mg and 23.60 mg, resp.). The pool size of chenodeoxycholic acid, plus its metabolites (α- and β-muricholic acids), was of the same magnitude in the two groups of animals. The daily production of cholic acid was decreased in phenobarbital-treated rats compared to control rats (2.12 mg and 7.24 mg, resp.). No significant difference was observed between the synthesis of chenodeoxycholic acid in the 2 groups of animals. The decreased production of cholic acid in phenobarbital-treated rats is in contrast to man where it is stimulated under these conditions. The results provide support for the view that various species and mammalian strains have differences in their ability to induce liver enzymes during drug treatment.

0127 ONTOGENESIS OF CYTOPLASMIC ORGANELLES IN RAT HEPATOCYTES AND THE EFFECTS OF PRENATAL PHENOBARBITAL ON ENDOPLASMIC RETICULUM DEVELOPMENT. *(E.)* Chedid, A.; Nair, V. (Michael Reese Hosp., Chicago, Ill.). *Dev. Biol.* 39(1):49-62, 1974.

The ontogenesis of liver cytoplasmic organelles was studied by electron microscopy in the rat from the first day of appearance of liver primordium to postnatal day 5. On gestation day 11, numerous ribosomes, a large Golgi complex, prominent centrioles, and a few mitochondria and microbodies were present. The free ribosomes decreased progressively to reach the adult appearance by day of birth. The centrioles, present in almost all cells on day 11 and 13 decreased progressively and were only occasionally seen by day 17 and thereafter. The mitochondria increased progressively in number and complexity. The Golgi complex, large and multiple during fetal life, became single, small, and inconspicuous after birth. The microbodies remained without significant changes throughout development, increased sharply at birth, and remained as such postnatally. Lysosomes, absent throughout prenatal life, appeared in large numbers at birth and acquired the adult pattern at that time. Rough endoplasmic reticulum became most prominent on gestation day 13. Electron dense material, seen for the first time within the rough endoplasmic reticulum cisternae on day 15, increased further until day 20 and disappeared at birth. Smooth endoplasmic reticulum, absent during the early fetal period, appeared for the first time gestation day 20. The number of cells containing smooth endoplasmic reticulum, as well as the amount of smooth endoplasmic reticulum, progressively increased. However, smooth endoplasmic reticulum distribution was still not comparable to that in the adult hepatocyte on postnatal day 5. Attempts to induce smooth endoplasmic reticulum formation prior to gestation day 20, its normal time of appearance, using phenobarbital as inducer, were unsuccessful.

0128 EFFECTS OF PHENOBARBITAL AND ETHANOL ON RAT LIVER ALDEHYDE DEHYDROGENASES. *(E.)* Tottmar, S. O. C.; Kiessling, K. H.; Forsling, M. (Inst. Zoophysiol., Univ. Uppsala, Sweden). *Acta Pharmacol. Toxicol. (Kbh.)* 35(4):270-276, 1974.

Liver microsomal aldehyde dehydrogenases and the drug-metabolizing system (the mixed function oxidase) were studied in Wistar rats treated with ethanol and phenobarbital. Phenobarbital treatment (100 mg/kg i.v. daily for 3 weeks) had no effect on the microsomal aldehyde dehydrogenase activity, whereas the mitochondrial aldehyde dehydrogenase activity was slightly decreased and the activities of ethylmorphine demethylase, NADPH-cytochrome *c* reductase and the concentration of cytochrome P-450 were increased 2 to 3-fold. Ethanol treatment (3 g/kg by gastric intubation for 3 weeks) caused no changes in the activities of the aldehyde dehydrogenases, and the drug metabolizing system was only slightly affected. Very little aldehyde dehydrogenase activity was found in the cytosol from livers of either control, ethanol-treated or phenobarbital-treated rats. Although the functional roles of the aldehyde dehydrogenases in the liver are largely unknown, it is conceivable that acetaldehyde formed from ethanol may exert harmful effects on different metabolic processes by acting as a competetive inhibitor of endogenous or dietary aldehydes.

0129 THE EFFECT OF ACUTE ETHANOL INGESTION ON IN VITRO METABOLISM OF CHOLINE AND ETHANOLAMINE DERIVATIVES IN RAT LIVER. *(E.)* Roberti, R.; Binaglia, L.; Michal, G.; Brunetti, M.; Porcellati, G. (Med. Sch., Univ. Perugia, Italy). *Biochem. Pharmacol.* 23(23):3289-3298, 1974.

The effect of acute ethanol ingestion (6 g/kg *via* gastric tube) on hepatic phospholipid metabolism was studied in fasting female Wistar rats. Neither lecithin nor phosphatidylethanolamine content changed significantly in liver during the 12-hr period following ethanol ingestion. The triglyceride content of the liver increased 3-fold. An increase of lecithin and phosphatidylethanolamine synthesis by the Kennedy pathway was found *in vitro* in the homogenates and microsomal fractions prepared from the ethanol-treated rats. A higher rate of conversion of phosphorylcholine and phosphorylethanolamine to cytidine 5'-diphosphate-choline and cytidine 5'-diphosphate-ethanolamine was also found in the experimental animals. The breakdown of cytidine 5'-diphosphate

choline to phosphorylcholine was noticeably decreased in the liver fractions of the ethanol-treated rats. No changes in the sequential methylation pathway for lecithin synthesis in the liver were observed after acute ethanol ingestion. The differing responses of the 2 pathways (Kennedy and stepwise methylation) to ethanol treatment could constitute an efficient means of controlling the availability of specific molecular species of lecithins for the lipoproteins to transport triglycerides to the plasma.

0130 AN EXPERIMENTAL MODEL OF ALCOHOL FEEDING AND LIVER INJURY IN THE BABOON. *(E.)* Lieber, C. S.; De Carli, L. M. (VA Hosp., Bronx, N.Y.). *J. Med. Primatol.* 3(3):153-163, 1974.

A model has been developed for the administration to the baboon of ethanol as part of a nutritionally adequate liquid diet. With this regimen, all animals maintained their weight. Liver morphology remained normal in the controls, which were fed a 60% carbohydrate diet. Isocaloric substitution of carbohydrate (50% of total calories) by ethanol resulted in the production of fatty liver in all 9 animals, while 4 also developed alcoholic hepatitis and 1 progressed to cirrhosis. Significantly increased activities of serum glutamic oxaloacetic transaminase were observed in all hepatitic animals. Inebriation and manifestation of dependence upon withdrawal of the diet were observed. Chemical alterations produced by ethanol at the fatty liver stage were characterized by striking triglyceride accumulation and enhanced activities of microsomal enzymes, including the microsomal ethanol oxidizing system. The ability to induce hepatitis and cirrhosis by prolonged ethanol ingestion in the absence of a deficient diet disproves the popular belief that an adequate diet can prevent cirrhosis in alcoholics. These studies also support the hypothesis that hepatitis and cirrhosis are preceeded by fatty liver.

0131 ETHANOL-INDUCED CHANGES IN METHIONINE METABOLISM IN RAT LIVER. *(E.)* Finkelstein, J. D.; Cello, J. P.; Kyle, W. E. (VA Hosp., Washington, D.C.). *Biochem. Biophys. Res. Commun.* 61(2): 475-481, 1974.

Male Sprague-Dawley rats (5/group) maintained on 3-10 day diets of 3.5, 26, or 55% casein were given daily isocaloric feeding (gastric intubation) of either ethanol (4.4 g/kg, 50% solution) or glucose (50% solution) to study ethanol-induced methionine metabolism changes in the liver. Ethanol administration had minimal effect on liver enzyme activities of rats fed 26 or 55% casein diets, with the only significant alteration being a 44-68% increase in s-adenosylmethionine synthetase specific activity for animals fed 26% casein as compared to glucose-fed rats on a 3.5% casein diet. Ethanol-treated rats on the 3.5% casein diet showed increases in s-adenosylmethionine synthetase, cystathionine synthase, and betaine-homocysteine methyltransferase activities of 56, 138, and 201%, resp., compared to controls. A 28% decrease in 5-methyltetrahydrofolate-homocysteine methyltransferase activity was also observed. Variation of alcohol administration from 24 hr to 10 days in 9 rats on 3.5% casein showed similar enzymatic activity patterns, with the 10-day patterns similar to the 3-day. The alcohol-treated rats gained weight at the same rate as controls on 3.5% casein. The results suggest a mechanism for increased dietary methionine (and choline) requirement in rats fed alcohol in combination with a protein-restricted diet. The observed enzyme changes represented a reversal of the normal adaptive response of the liver to protein-restriction, and the resultant impairment in methionine conservation could explain alcohol-induced increases in dietary lipotrope requirement.

0132 LIPID PEROXIDATION IN CHRONIC ETHANOL TREATED RATS: IN VITRO UNCOUPLING OF PEROXIDATION FROM REDUCED NICOTINE ADENOSINE DINUCLEOTIDE PHOSPHATE OXIDATION. *(E.)* Koes, M.; Ward, T.; Pennington, S. (Cancer Res. Cent., Columbia, Mo.). *Lipids* 9(11):899-904, 1974.

In vivo and *in vitro* experiments with male and female Holtzman rats verified that chronic ethanol exposure (3 g/100 g/day) for 3 weeks (stomach tube) induces microsomal ethanol oxidizing system (MEOS) components and that lipid peroxidation is enhanced in ethanol-treated animals. Clearance data (blood ethanol level relative to an 8-hr sample) for 5 controls and 5 chronic ethanol-treated rats intoxicated with 6 g/kg ethanol showed that the latter cleared about 90% of the 8-hr ethanol value after 16 hr compared to a 60% 16-hr clearance for controls. Rats pretreated with pyrazole had a 16-hr blood ethanol level greater than the 8-hr level when given the same intoxicating dose in comparison to rats pretreated with ethanol and pyrazole which cleared 20% of the 8-hr ethanol level. Both groups had comparable pyrazole levels and thus equal metabolism rates. *In vitro* addition of reduced NADPH (0.25 μM) to isolated rat microsomes caused 200% malonaldehyde elevations. The appearance of peroxidized materials for ethanol-treated rats, following the addition of substrate for NADPH oxidase, suggests that NADPH oxidation lies near the rate determining step for peroxidized lipid formation in microsomal preparations. Azide ion addition to microsomes of control and chronic ethanol-treated rats did not greatly influence NADPH oxidase activity, although malonaldehyde formation was depressed 90% in controls and 65% in ethanol-treated animals. It is suggested that lipid peroxidation did not appear to be irreversibly linked to NADPH oxidation.

0133 EFFECTS OF PYRAZOLE ON ETHANOL-INDUCED CHANGES IN HEPATIC TRIGLYCERIDE AND GLYCEROL-1-PHOSPHATE CONTENTS AND ON ESTERIFIED AND NON-ESTERIFIED FATTY ACIDS IN THE BLOOD. *(E.)* Estler, C. J. (Inst. Pharmacol., Univ. Erlangen-Nuremberg, Erlangen, Germany). *Res. Exp. Med. (Berl.)* 163(2):95-100, 1974.

The effects of ethanol and/or pyrazole administration to 3 groups of female NMRI mice on esterified and non-esterified fatty acids in the blood and on the

amount of triglycerides, glycerol-1-phosphate, and dihydroxyacetone phosphate in the liver were examined following acute ethanol administration. Mice receiving 4 mg/g ethanol i.v. showed rapid accumulation (within 2 hr) of triglycerides in the liver, with levels increasing from 12.97 to 26.89 μval/ml. Glycerol-1-phosphate increased from o.23 to 0.88 M/g, while dihydroxyacetone phosphate did not show a statistically significant decrease. Nonesterified fatty acids (blood) were 0.002 μM/g; these were increased 0.05 μval/ml over control values and esterified fatty acids (blood) decreased by 1.45 μval/ml. Mice receiving 340 μg/g pyrazole i.p. 2.25 hr presacrifice did not show significant differences in these blood and liver parameters. Mice pretreated with with 340 μg/g pyrazole i.p. 15 min prior to ethanol treatment (4 mg/g, i.v.) showed in comparison to pyrazole-only mice a 0.05 μval/ml increase in nonesterified fatty acids, increases of 2.42 μval/ml and 13 mg/g for esterified fatty acids and triglycerides, resp., and decreases in glycerol-1-phosphate and dihydroxyacetone phosphate from 0.22 μM/g and 0.027 μM/g, resp. to 0.16 and 0.021 μM/g, resp. Alcohol-induced lipolysis appeared to be caused by ethanol alone since the nonesterified fatty acids in the blood also rose in pyrazole-pretreated animals in which the formation of acetaldehyde should be negligible. It was concluded that the initial phase of hepatic steatosis produced by intoxicating doses of ethanol is due to factors other than the alteration of the liver cell's redox state.

0134 HEPATIC REDOX STATE: ATTENTUATION [SIC!] OF THE ACUTE EFFECTS OF ETHANOL INDUCED BY CHRONIC ETHANOL CONSUMPTION. *(E.)* Domschke, S.; Domschke, W.; Lieber, C. S. (Dep. Med., Univ. Erlangen-Nuremberg, Erlangen, Germany). *Life Sci.* 15(7):1327-1334, 1974.

The effects of an acute dose of a diet containing ethanol (3 g/kg) on hepatic redox state was compared in rats fed ethanol for 25 days and in their littermates given isocaloric carbohydrate. In both groups, cytoplasmic and mitochondrial redox states of pyridine nucleotides shifted to a more reduced level (decreased NADP/NADPH ratio), but the changes were much less extensive in rats chronically fed ethanol. This metabolic adaption may reflect the oxidation of ethanol by a pathway not involving alcohol dehydrogenase, such as the microsomal ethanol oxidizing system, which increases in activity after chronic ethanol ingestion.

0135 ETHANOL AND DRUG METABOLISM IN MOUSE LIVER MICROSOMES SUBSEQUENT TO LIPID PEROXIDATION-INDUCED DESTRUCTION OF CYTOCHROME P-450. *(E.)* Vastis, K. P.; Kowalchyk, J. A.; Schulman, M. P. (Univ. Illinois Coll. Med., Chicago). *Biochem. Biophys. Res. Commun.* 61(1):258-264, 1974.

In vitro studies were undertaken to determine the effect of NADPH-induced peroxidation on the capacity of microsomes to catalyze the NADPH-dependent oxidation of ethanol. Preincubation of mouse liver microsomes with NADPH resulted in malondialdehyde formation, destruction of cytochrome P-450, and decreased rates of aniline hydroxylation and N-demethylation of aminopyrine and ethylmorphine. These phenomena were more pronounced in phosphate than in Tris buffer. No reduction in rates of NADPH-linked oxidation of ethanol or in the activities of NADPH oxidase and NADPH-cytochrome c reductase was observed. While addition of EDTA to preincubation mixtures prevented lipid peroxidation, loss of cytochrome P-450, and inactivation of the drug-metabolizing capacity of microsomes, it did not alter ethanol oxidation rates and the activities of NADPH oxidase and NADPH-cytochrome c reductase. These findings argue against the involvement of cytochrome P-450 in the microsomal ethanol-oxidizing system.

0136 THE EFFECTS OF SUBSTRATES OF MIXED FUNCTION OXIDASE ON ETHANOL OXIDATION IN RAT LIVER MICROSOMES. *(E.)* Hildebrandt, A. G.; Speck, M.; Roots, I. (Inst. Clin. Pharmacol., Free Univ. Berlin, Germany). *Naunyn Schmiedebergs Arch. Pharmacol.* 281(4):371-382, 1974.

The effects of aminopyrine and ethylmorphine on reduced NADPH and oxygen-dependent ethanol oxidation in rat liver microsomes were studied in male Sprague-Dawley rats. Animals were given 80 mg/kg body weight of phenobarbital 72, 60, 48, and 24 hr prior to decapitation for induction of microsomal enzymes. Studies of the rate of formation of acetaldehyde from 50 mM ethanol with isolated microsomal suspensions revealed that in the presence of excess catalase both aminopyrine (8 mM) and ethylmorphine (8 mM) stimulated ethanol oxidation (25 and 26%, resp.). In the absence of catalase, the control activity of the microsomal suspensions was decreased by 39%, and the presence of either substrate at the above concentrations inhibited the rate of acetaldehyde formation by 19 and 38%, resp. The addition of azide to the microsomes in the absence of catalase resulted in a 72% inhibition of acetaldehyde formation which was potentiated by aminopyrine and ethylmorphine to 75 and 87%, resp. These effects were not observed when methanol was used as a substrate, demonstrating that the inhibitions were specific for microsomal ethanol oxidation. Excess catalase plus aminopyrine resulted in strong inhibition of acetaldehyde formation at low ethanol concentrations (below about 20 mM) and a stimulation of the aldehyde's formation at high concentrations (30-140 mM) of ethanol. Lineweaver-Burk plots of acetaldehyde formation *versus* ethanol concentration indicated the simultaneous action of 2 enzymes functional in ethanol oxidation. The results are consistent with the observation that microsomal ethanol oxidation cannot be accounted for solely by catalase activity and provide evidence for the existence of a microsomal ethanol oxidizing enzyme system.

0137 REDUCED GLUCAGON INDUCTION OF TYROSINE AMINOTRANSFERASE IN PERFUSED LIVERS AFTER ETHANOL TREATMENT. *(E.)* Morland, J. (Univ. Oslo, Norway). *Biochem. Pharmacol.* 23(23):3239-3246, 1974.

Male Wistar rats (250–290 g body weight) were given ethanol for 7 weeks to provide either 22 or 35% of the total calories while sucrose replaced ethanol isocalorically in two respective control groups. Ethanol treatment did not influence basal hepatic tyrosine aminotransferase activity. When the livers were isolated and perfused in the presence of glucagon for 3 hr, the subsequent increase of tyrosine aminotransferase activity was reduced to approximately 70% in livers from animals treated with ethanol. This reduction was probably caused by reduced enzyme synthesis, since no action could be found on enzyme activation, degradation or leakage. Cyclic AMP levels in the livers were not affected by previous ethanol treatment. The reduced tyrosine aminotransferase synthesis seems therefore to be independent of changes in the hormone receptor or in the accumulation of cyclic AMP in the liver after glucagon. General protein synthesis, as determined by the rate of incorporation of isotopically labeled amino acid precursor, was not influenced by ethanol treatment in livers perfused with glucagon.

0138 EFFECT OF AN INTOXICATING DOSE OF ETHANOL OR ISOPROPANOL ON THE INCORPORATION OF [1-^{14}C] PALMITATE INTO RAT LIVER AND BLOOD LIPIDS. *(E.)* Beauge, F.; Clement, M.; Nordmann, J.; Nordmann, R. (INSERM, Salpetriere Hosp., Paris, France). *Biomedecine* 21(9):386-389, 1974.

The effect of acute ethanol or isopropanol administration (6 g/kg body weight by stomach intubation) on [1-^{14}C] palmitate incorporation into hepatic and blood plasma triglycerides and phospholipids was studied in fasting female Wistar rats. Ethanol produced no significant alteration in the specific activity or concentration of liver plasma triglycerides or phospholipids compared to controls. Isopropanol also had no effect on liver and plasma triglyceride and phospholipid concentration. It did, however, produce a 50% decrease in hepatic triglyceride and phospholipid specific activity and a 90% decrease in serum specific activity. These results indicate that isopropanol, but not ethanol, inhibits release of triglycerides and phospholipids from the liver into serum. Thus, impaired hepatic triglyceride secretion does not appear to be a factor in the development of ethanol-induced fatty liver.

0139 BIOSYNTHETIC PATHWAYS AND TURNOVER OF INDIVIDUAL BILIARY PHOSPHATIDYLCHOLINES DURING METABOLISM OF [1,1-$^{2}H_2$] ETHANOL IN THE RAT. *(E.)* Curstedt, T.; Sjovall, J. (Karolinska Inst., Stockholm, Sweden). *Biochim. Biophys. Acta* 369(2):173-195, 1974.

Incorporation of deuterium into different positions of individual molecular species of biliary phosphatidylcholines was determined in bile fistula rats given i.p. [1,1-$^{2}H_2$]ethanol at a constant rate for 24 hr. The distribution of deuterium between the different positions of the glycerol moiety was the same for all major molecular species. The deuterium excess in phosphatidylcholine molecules synthesized *de novo* was calculated; the values obtained were the same for all molecular species analyzed and remained constant throughout the experiment. The deuterium excess was 35–55 atom % at C-2, 18–20 atom % at C-3 and 8–10 atom % at C-1. Administration of glucose and fructose decreased the deuterium excess at C-3 to 13–15 and 2–4 atom %, resp., but did not change it at C-1 or C-2. It was concluded that all molecular species shared the same precursor pools of dihydroxyacetone phosphate and pyridine nucleotide, and that these pools were closely coupled to ethanol oxidation. The deuterium labeling at C-3 compared with that of potential precursors, as well as the effects of glucose and fructose on the labeling at C-3, indicated that at least 65–70% of the glycerol moieties of the phosphatidylcholines were formed from malate *via* a gluconeogenetic pathway. About 20–30% of the palmitoyl residues were synthesized *de novo*. This value increased to 31–64% when glucose was administered and to 79–100% when fructose was given. This change of origin was accompanied by a marked increase of the 1-palmitoyl-2-palmitoleoyl, 1-palmitoyl-2-oleoyl, and 1-palmitoleoyl-2-oleoyl species, and a decrease of species having linoleoyl or arachidonoyl residues at C-2. The deuterium excess of the NADPH pool used in palmitic acid synthesis was calculated to be 5–10 atom %. The rates of deuterium incorporation indicated the presence of at least two pools of phosphatidylcholines with widely different turnover. The apparent half-life times of the glycerol moiety of molecular species in the pool having a rapid turnover varied between about 1 hr (1,2-dipalmitoyl species) and about 8 hr (1-stearoyl-2-linoleoyl species). The apparent half-life times of 1-palmitoyl residues in different molecular species were about 4 and 10 hr in rats given glucose and fructose, resp. The values were the same for all species studied, indicating that labeled palmitoyl residues in different molecular species were derived from the same palmitic acid pool.

0140 EFFECT OF ETHANOL AND FRUCTOSE ON GALACTOSE ELIMINATION IN RAT. *(E.)* Keiding, S. (Rigshosp., Copenhagen, Denmark). *Scand. J. Clin. Lab. Invest.* 34(1):91-96, 1974.

The effect of ethanol and fructose on galactose elimination has been studied in rats. Nephrectomized female Wistar rats received a bolus of galactose (0.04-0.4 mM) *via* the jugular vein followed by continuous infusion (40–180 μM/hr) over a 60 min period. Either ethanol or fructose were given as a single i.v. bolus (1.0 and 1.1 mM, resp.) immediately before the galactose bolus followed by continuous infusion (0.76 and 1.2 mM/hr, resp.) mixed with the galactose. Arterial ethanol and fructose concentrations during infusion were 7 and 6 mM/liter, resp. The galactose elimination capacity, defined as the elimination rate at blood galactose concentrations between 2 and 7 mM/liter, during ethanol infusion was, on the average, 80 μM/hr per whole animal, or two-thirds the value observed in comparable experiments without ethanol. Fructose did not change the galactose elimination capacity. The galactose elimination during ethanol infusion could be described by Michaelis-Menten kenetics. The calculated V_{max} was significantly lower than that obtained in experiments without

ethanol. The pattern of the metabolites observed in liver tissue is compatible with an inhibitory effect of ehtanol on UDP-glucose-4-epimerase, but with the phosphorylation of galactose by galactokinase as the rate-limiting step, inhibited by accumulation of galactose-1-phosphate. The galactose-1-phosphate concentration observed indicates a considerably smaller K_I for the inhibition of galactokinase by galactose-1-phosphate towards galactose than that observed by *in vitro* studies.

0141 INCREASE IN HEPATIC NAD LEVEL—ITS EFFECT ON THE REDOX STATE AND ON ETHANOL AND ACETALDEHYDE METABOLISM. *(E.)* Erikson, C. J. P. (Res. Lab., State Alcohol Monop., Helsinki, Finland). *FEBS Lett.* 40(2):317-20, 1974.

Studies were conducted in male Wistar rats to determine the effects of liver NAD levels on ethanol and acetaldehyde metabolism. Experimental rats received an i.p. injection of nicotinamide (500 mg/kg) followed 8 hr later by i.p. ethanol (1.18 g/kg). Ethanol caused about a 50% increase in liver NADH and a slight decrease in NAD in nicotinamide-pretreated rats, values insignificantly different from those of non-nicotinamide-pretreated controls. Both total and free liver NADH:NAD ratios were increased 4-fold following ethanol injection in both experimental and control animals. The cytoplasmic free NADH:NAD ratio as calculated from the lactate:pyruvate ratio showed no response to increased total NAD either before or during ethanol metabolism. These results could be due either to accumulation of NAD in some compartment other than the cytosol, or to release of bound NADH in the cytosol. No effect on ethanol elimination in rats was obtained by increasing the hepatic NAD level with nicotinamide. However, blood acetaldehyde concentration was significantly lowered in nicotinamide-pretreated animals. The decreased acetaldehyde levels could be due to stimulation of hepatic acetaldehyde oxidation by NAD or to the decreased ethanol oxidation state shown to exist in these rats.

0142 TRANSFER OF DEUTERIUM FROM [1-$^2H_2$]ETHANOL TO KREBS CYCLE AND RELATED ACIDS OF RAT LIVER IN VIVO. *(E.)* Cronholm, T.; Matern, H.; Matern, S.; Sjovall, J. (Karolinska Inst., Stockholm, Sweden). *Eur. J. Biochem* 48(1):71-80, 1974.

The utilization in intermediary metabolism of hydrogens derived from C-1 in ethanol was studied in male Sprague-Dawley rats following 2 i.p. or i.v. injections of [1-$^2H_2$]ethanol (1.0 g/kg body weight) 2 hr apart. A gas chromatographic-mass spectrometric method was developed and used to measure the deuterium content in individual positions of lactate, malate, 3-hydroxybutyrate, citrate, succinate and fumarate isolated from freeze-clamped livers of rats metabolizing [1-$^2H_2$]ethanol (95 atoms% ^2H excess) at a maximal rate. A steady state of ^2H-labeling was reached within a few minutes. 3-Hydroxybutyrate contained only 1-2 atoms% of deuterium which was interpreted to indicate that intramitochondrial NADH was essentially unlabeled due to exchange with protons in the NADH dehydrogenase reaction. A low labeling of lactate, 17 atoms%, indicated that the exchange of hydrogens in cytosolic NADH with unlabeled substrates and water was 4 times higher than the rate of ethanol oxidation. Since intramitochondrial oxidation of NADH is not rate limiting, the presence of labeled NADH in the cytosol may indicate that transfer of hydrogen through the mitochondrial membrane is a rate-limiting step. As expected from the reversible reactions catalyzed by fumarate hydratase and malate dehydrogenase the deuterium excess at C-2 and C-3 of malate and fumarate were about the same (20 atoms%). Citrate had a deuterium excess of about 9 atoms%, assuming that only 1 hydrogen was labeled. Succinate contained very little deuterium. The labeling of citrate may thus be due either to incorporation of oxaloacetate labeled at C-3 or to labeling of the same hydrogen in a reaction catalyzed by isocitrate dehydrogenase. The results indicate that both malate and isocitrate are potential sources of labeled NADPH during oxidation of [1-$^2H_2$]ethanol, and that these 2 routes are not completely equilibrated under the experimental conditions.

0143 UTILIZATION OF THE CARBON AND HYDROGEN ATOMS OF ETHANOL IN THE BIOSYNTHESIS OF STEROIDS AND BILE ACIDS. *(E.)* Cronholm, T.; Burlingame, A. L.; Sjoval, J. (Karolinska Inst., Stockholm, Sweden). *Eur. J. Biochem.* 49(3):497-510, 1974.

Male Sprague-Dawley rats with bile fistulas were given i.p. injections of [1-$^2H_2$]ethanol, [2-$^2H_3$]ethanol and [1-^{13}C,1-$^2H_2$]ethanol (10% solutions, 0.5-1.5 ml hourly for 23-24 hr) under conditions ensuring a maximal rate of oxidation. Cholesterol, cholic acid, chenodeoxycholic acid and 3β,11β,21-trihydroxy-6α-pregnan-20-one were isolated from bile collected at intervals during the experiment. The fraction of molecules containing different numbers of heavy isotope atoms was measured by gas chromatography, mass spectrometry, and computer techniques. The results were used to determine the origin of the cholesterol pools used as precursors for the different compounds isolated from bile. The bile acids were found to be formed from a cholesterol pool, the turnover of which was about 5 times more rapid than that of the cholesterol pool used for biliary excretion. The cholesterol pools used for biliary excretion and as corticosterone precursor seemed to be in equilibrium. The acetyl groups derived from ethanol were introduced into the steroids without exchange of hydrogens at C-2, and the acetyl-coenzyme A (CoA) pool used as precursor did not exchange with acetyl groups in, e.g., citrate or fatty acids. A difference in incorporation of [2-$^2H_3$]acetyl groups and [1-^{13}C] acetyl groups was interpreted to be due to an isotope effect. Ethanol contributed about 45% of the acetyl-CoA pool used as precursor of cholesterol and bile acids. Deuterium from [1-$^2H_2$]ethanol was incorporated in many positions of all the steroids. If it is assumed that the incorporation is mediated by NADPH, the deuterium excess of the hydrogens introduced was about 10 atoms%. The contribution *via* different pathways of an important part of the hydrogen and carbon atoms in cholesterol, bile acids

and corticosteroids provides possible mechanisms by which ethanol may affect the biosynthesis and metabolism of steroids.

0144 EFFECT OF 1,3-BUTANEDIOL ON HEPATIC FATTY ACID AND METABOLITE LEVELS IN THE RAT. *(E.)* Romsos, D. R.; Belo, P. S.; Leveille, G. A. (Dep. Food Sci. Hum. Nutr., Michigan State Univ., East Lansing). *J. Nutr.* 104(11):1438-1445, 1974.

The effect of 1,3-butanediol on hepatic fatty acid synthesis and metabolite levels was examined in male Sprague-Dawley rats. Hepatic fatty acid synthesis, as estimated by histochemical and biochemical techniques, was depressed in rats fed 1,3-butanediol. An i.p. injection of 1,3-butanediol (800 mg) increased blood glucose levels but did not affect hepatic fatty acid synthesis. Addition of 10 mM 1,3-butanediol to the incubation buffer depressed glucose, but not acetate, conversion to fatty acids by rat liver slices. Methylene blue inhibition of hepatic fatty acid synthesis was partially reversed by the addition of 1,3-butanediol to the incubation buffer. Hepatic β-hydroxybutyrate levels and the β-hydroxybutyrate: acetoacetate ratio were increased when 1,3-butanediol was fed (18 or 36% dietary energy). Lactate and pyruvate levels were lower in freeze-clamped liver preparations from rats fed 1,3-butanediol than those observed in control animals. Further, the lactate:pyruvate ratio was increased, suggesting that the hepatic cytoplasmic NADH:NAD+ ratio was increased. Hepatic long-chain acyl coenzyme A (CoA) levels were also increased when 1,3-butanediol was fed. It is suggested that the shift in the cytoplasmic redox state and the increase in hepatic long-chain acyl CoA levels are involved in the decrease in hepatic fatty acid synthesis observed when rats are fed 1,3-butanediol.

0145 BIOCHEMICAL CHANGES INDUCED BY ETHYL ACETATE IN BLOOD AND LIVER OF RAT. *(E.)* Seth, P. K.; Srivastava, S. P. (Ind. Toxicol. Res. Cent., Lucknow, India). *Bull. Environ. Contam. Toxicol.* 12(5):612-616, 1974.

The effects of ethyl acetate (1.0 mg/kg/day for 8 days) on blood and liver pyruvic acid content and on hepatic enzyme activity were studied in 10 male albino rats. Both liver and blood pyruvate concentration increased about 2-fold gradually from the 1st to the 7th day of ethyl acetate injection. Associated with this were both a decrease in liver glycogen from 3.98 to 2.21 µg/g wet weight and an increase in both blood (9.20 to 17.38 mg%) and liver 121.00 to 283.40 µg/g wet weight) lactic acid, also changes of about 2-fold. The increased blood and hepatic pyruvate levels in ethyl acetate-treated rats may have been due to an increase rate of glycolysis since hepatic glucose-6-phosphatase activity was unchanged. Adenosine triphosphatase, 5'-nucleotidase and acid phosphatase activities were not significantly changed, suggesting that hydrolysis of phosphate esters is generally unaffected by ethyl acetate.

0146 EFFECTS OF TETRACYCLINE ON ULTRASTRUCTURE AND LIPOPROTEIN SECRETION IN THE RAT HEPATOCYTE. *(E.)* Gray, J. E.; Weaver, R. N.; Skinner, P.; Matthews, J.; Day, C. E.; Stern, K. (Res. Lab. Upjohn Co., Kalamazoo, Mich.). *Toxicol. Appl. Pharmacol.* 30(2):317-332, 1974.

Ultrastructural and biochemical studies were conducted to definitively characterize the progressive morphological changes which occur at near-maximum tolerated levels of i.p. administered tetracycline in Sprague-Dawley rats. Fat droplets, 0.1-0.5 µm, accumulated in the space of Disse of hepatocytes in rats treated with 300 mg/kg. This change occurred in both pregnant and unbred females and in males but was not observed in similar rats treated with 100 mg/kg tetracycline which showed typical steatosis. Ultrastructural changes in mitochondria, and rough and smooth endoplasmic reticulum were similar to those previously reported. Uptake of i.v. administered palmitic [9-10-^{3}H]acid in rats treated with 300 mg/kg was not appreciably impaired. One hour after an injection of 300 mg/kg, the antibiotic concentration was 10 times greater in the liver than in the serum. From 1-6 hr the concentrations increased from roughly 300 to 800 µg/g of liver. In rats treated with 100 mg/kg, no concentrations above 200 µg/g were observed during a 48-hr postinjection period. Under *in vitro* conditions, tetracycline complexed with lipoprotein in a precipitate when a concentration greater than 200 µg/ml was obtained in rat blood serum. In the hepatocytes of rats treated with 300 mg/kg it was presumed that as the antibiotic concentration rose above 200 µg/g nascent very low density lipoprotein complexed rapidly with it. The complexed lipoprotein was channeled in dilated smooth endoplasmic reticulum in the manner of liposomes to the sinusoidal surface and discharged from vesicles into the space of Disse. The plasma membrane of the hepatocyte showed loss of microvilli and surface distortion. The space of Disse became distended with insoluble fat droplets from 6 hr to 3 days after injection, due to the passage of fine osmiophilic droplets or liposomes from the sinusoidal surface of the hepatocyte.

0147 ACUTE HEPATOTOXIC EFFECTS OF MIREX IN THE RAT. *(E.)* Kendall, M. W. (Univ. Mississippi Sch. Med., Jackson). *Bull. Environ. Contam. Toxicol.* 12(5):617-620, 1974.

Light microscopic studies were conducted to study the acute hepatotoxic effects of Mirex, a chlorinated polyhalogen insecticide. Female Sherman rats received either a single lethal dose for 50% survival of Mirex (365 mg/kg) by i.p. injection or were fed *ad libitum* a total of 365 mg/kg Mirex in their diet over a 12 day period. I.p. administration produced apparent glycogen depletion with hepatocyte enlargement, periportal liposis with central necrosis, and infrequent areas of surface necrosis in livers after 12 days. Oral feeding produced all these changes except for surface necrosis. Clinically, rats fed p.o. exhibited lethargy, loss of hair and weight, and tremor and diarrhea, all previously described symptoms of Mirex toxicity. The pattern of periportal

liposis seems to be fairly specific for Mirex toxicity in rats.

0148 CATION MOVEMENT DURING CARBON DISULPHIDE-INDUCED LIVER INJURY. *(E.)* Butler, W. H.; Hempsall, V.; Magos, L. (Med. Res. Counc. Lab., Carshalton, England). *J. Pathol.* 113(1):53-60, 1974.

The changes in liver water content and levels of Na+ and K+ occurring after acute exposure to carbon disulfide (4 mg/liter for 4 hr) were studied over a 7-day period in phenobarbital-pretreated starved male albino rats. Compared to controls, hepatocyte water content showed a rapid increase, reaching a maximum of about 2.8 kg/kg dry weight at 12 hr after carbon disulfide exposure and returning to normal after 48 hr. Associated with this and following the same time course, there was an increase in Na+ and K+ content related to liver dry weight. The concentration of Na+ and K+, when expressed in relation to the total liver water, did not change. It was suggested that carbon disulfide causes dissolution of the smooth endoplasmic reticulum, thus forming an osmotically active species resulting in the increased water content. Normal Na+ and K+ concentrations are maintained by an intact Na+ pump. During recovery from carbon disulfide, the damaged cell membrane is possibly included in autophagic vacuoles and is no longer available to the cell.

0149 FATE OF ARSENIC AFTER PARENTERAL ADMINISTRATION TO RATS, WITH PARTICULAR REFERENCE TO EXCRETION VIA BILE. *(E.)* Cikrt, M.; Bencko, V. (Fac. Med. Hyg., Charles Univ. Prague, Czechoslovakia). *J. Hyg. Epidemiol. Microbiol. Immunol. (Praha)* 18(2):129-136, 1974.

Biliary arsenic excretion was studied in fasting female Wistar rats for 24 hr following i.v. administration of ^{74}As-labeled sodium arsenate or arsenite (100 µg As). Total 24 hr biliary arsenic excretion was 10.82% and 1.42% of the administered doses of arsenite and arsenate, resp. The highest rate of excretion occurred during the 1st 15 min after injection in both cases. Urinary arsenic excretion over the 24-hr period was significantly lower for arsenite (6.00%) than for arsenate (29.39%). Arsenic content as determined by chromatographic analysis of tissue homogenates was higher in liver and kidneys after arsenite injection and in urine and erythrocytes after arsenate administration. Significant differences between the 2 valency forms were not found in either the wall or the content of the different levels of the gastrointestinal tract. It was thus concluded that the biliary excretion of arsenic is significant, especially following arsenite administration. The differences in the rate of excretion of the 2 valency forms may be due to differences in the binding pattern to the different bile components.

0150 MECHANISMS OF ACUTE HEPATIC TOXICITY: CHLOROFORM, HALOTHANE, AND GLUTATHIONE. *(E.)* Brown, Jr., B. R.; Sipes, I. G.; Sagalyn, A. M. (Univ. Arizona Coll. Med., Tucson). *Anesthesiology* 41(6)554-561, 1974.

Hepatotoxic response to chloroform and halothane anesthesia was studied in male Sprague-Dawley rats pretreated with 1 mg/ml p.o. phenobarbital. The effect of diethyl maleate pretreatment, particularly on glutathione level was also investigated. Hepatic glutathione content in phenobarbital-pretreated animals was decreased 70% after 2 hr of exposure to 0.5% chloroform anesthesia and 83% after 2 hr exposure to 1% chloroform. Glutathione concentrations were not altered following chloroform anesthetization of controls not receiving phenobarbitol pretreatment. Neither controls nor pretreated animals showed glutathione changes after treatment with 1% halothane. Mortality rates at 18 hr were 20% for phenobarbital-pretreated rats anesthetized with 0.5% chloroform and 80% with 1.0% chloroform. No deaths occurred in controls receiving chloroform or in any of the animals receiving halothane. Marked destruction of microsomal electron transfer components was observed in the phenobarbital-pretreated animals receiving chloroform. Chloroform in the phenobarbital pretreated animals caused marked destruction of cytochrome P-450 and reduced nicotinamide adenine dinucleotide phosphate cytochrome c reductase. These animals also exhibited increased amounts of microsomal cytochrome b_5. Diethyl maleate administration produced rapid reduction in glutathione content in all groups of animals. Two hr after i.p. administration of 0.6 ml/kg, the mean glutathione level in rats was 0.474 µg/mg liver as compared to a control value of 2.15 µg/mg liver. Chloroform anesthesia (0.5%) in the presence of glutathione depletion resulted in increased diene conjugates and cytochrome P-450 destruction in animals not receiving phenobarbital pretreatment. Starvation (48 hr) was also found to increase the sensitivity of the liver to toxic actions of chloroform. *In vitro* experiments revealed that microsomal protein from livers of phenobarbital-pretreated animals bound chloroform metabolites to a much greater extent than did microsomal protein from non-induced livers. Addition of glutathione (2mM) markedly inhibited the binding of these metabolites by phenobarbitol-induced microsomes and reduced binding in noninduced microsomes.

0151 INTERACTION OF VOLATILE ANESTHETICS WITH RAT HEPATIC MICROSOMAL CYTOCHROME P-450. *(E.)* Takahashi, S.; Shigematsu, A.; Furukawa, T. (Fac. Med., Kyushu Univ., Fukuoka, Japan). *Anesthesiology* 41(4):375-379, 1974.

The effects of halothane, methoxyflurane, and enflurane on hepatic microsomal suspensions from adult male Wistar rats were spectrophotometrically studied to investigate their interaction with cytochrome P-450. Hepatic microsomal enzymes were induced by i.p. phenobarbital pretreatments (80 mg/kg/day, 4 days). Addition of each of the anesthetics (about 6 mM) to the suspensions caused the appearance of a trough at 420 mµ and an absorption band at 385 mµ (type I spectral change), indicative of an interaction between cytochrome P-450 and the anesthetics. Diethyl ether had no effect. The 3 anesthetics competitively

inhibited the spectral change produced by addition of 1.0 mM hexobarbital (type I substrate), while the spectral change (trough at 393 mμ and absorption band at 430 mμ) caused by aniline (type II substrate) was increased in amplitude by these volatile anesthetics. N-demethylase activity of aminopyrine (5 mM; type I substrate) was inhibited and the hydroxylase activity of aniline enhanced by the anesthetics. The biotransformation of all 3 anesthetics appeared to be dependent on interaction with cytochrome P-450. Furthermore, induction of microsomal mixed-function oxidases appeared to change the rates of metabolism of each of the anesthetics.

0152 FURTHER STUDIES CONCERNING THE EFFECTS OF CLOFIBRATE ON RESPIRATION AND OXIDATIVE PHOSPHORYLATION OF RAT LIVER MITOCHONDRIA. *(E.)* Mackerer, C. R.; Haettinger, J. R. (Searle Lab., Chicago, Ill.). *Biochem. Pharmacol.* 23(23):3331-3345, 1974.

The hypolipidemic agent, clofibrate (>0.1 μM/mg protein) administered *in vitro*, inhibited rat liver mitochondrial respiration at 2 sites within the respiratory chain. One site was between the interaction of NADH with NADH dehydrogenase and the point at which electrons from succinate oxidation enter the electron transport chain; another, less sensitive site, was between the interaction of succinate with succinate dehydrogenase and cytochrome c. In addition to these specific sites, clofibrate inhibited respiration by causing a depletion of pyridine nucleotides that was accompanied or followed by large-amplitude, non-energy-linked swelling. Clofibrate uncoupled oxidative phosphorylation at coupling sites II and III but not at site I. The concentrations required to cause loss of pyridine nucleotides were lower than those required to inhibit at the specific sites. *p*-Chlorophenoxyisobutyrate (CPIB) also inhibited succinate and β-hydroxybutyrate-linked respiration and uncoupled oxidative phosphorylation but at much higher concentrations (50% inhibition of β-hydroxybutyrate oxidation at about 3.7 μM/mg of protein) than were required of clofibrate (50% inhibition of β-hydroxybutyrate oxidation at about 0.17 μM/mg of protein). Clofibrate administration to rats (100 and 300 mg/kg p.o. daily for 1 week) lowered serum lipid levels and increased the liver size, the amount of mitochondrial protein/g of liver, and the oxygen consumption of liver slices. However, mitochondria, isolated from livers of the treated rats, respired normally. A single administration of clofibrate (100 or 300 mg/kg, p.o.) did not affect liver slice respiration.

0153 SYNERGISM BETWEEN AMMONIA AND FATTY ACIDS IN THE PRODUCTION OF COMA: IMPLICATIONS FOR HEPATIC COMA. *(E.)* Zieve, F. J.; Zieve, L.; Doizaki, W. M.; Gilsdorf, R. B. (Minneapolis VA Hosp., Minnesota). *J. Pharmacol. Exp. Ther.* 191(1): 10-16, 1974.

Male Holtzman rats and mongrel cats were used to study the synergistic action of ammonia and fatty acids in producing coma. An i.p. dose of 1.45 mM NH_4^+ caused coma in 50% of rats receiving such treatment, and a 1.7 mM dose produced coma in 100% of the animals. The maximum dose of NH_4^+ not producing coma in any animal was 1.2 mM i.p. The addition of 0.4 mM octanoate (i.p.) lowered the coma-producing NH_4^+ dose (in 50% of the rats) to 0.69 mM. Coma incidence rose from 0-100% when the maximum subcoma doses of NH_4^+ (1.2 mM i.p.) and octanoate (0.4 mM i.p.) were given simultaneously. A converse experiment with rats showed similar effects, with 1.0 mM NH_4^+ (i.p.) lowering the octanoate dose required for 50% coma incidence from 0.48 to 0.31 mM (i.p.). Measurements of blood ammonia and plasma free fatty acid levels in 136 normal rats receiving various combinations of ammonium chloride and octanoate revealed that coma was produced by ammonium chloride alone at blood ammonia nitrogen concentrations of approximately 2200 μg/100 ml and by octanoate alone at concentrations of approximately 2800 nM/ml. At a Dole free fatty acid concentration of 700 nM/ml, coma occurred when blood ammonia was 1000 μg/100 ml. Similar measurements in cats receiving i.v. combinations of ammonium acetate and octanoate showed almost identical findings. Subcoma doses of representative short, medium, and long chain fatty acids injected i.p. into rats just prior to injection of 1.0 mM ammonium chloride also resulted in the production of coma, with the average blood ammonia rising by at least 1000 μg/100 ml. The longer chain fatty acids were actually more potent than octanoate in this respect. Rats and cats in hepatic coma produced by portacaval shunt and ligation of the hepatic artery showed blood ammonias varying between 900 and 2500 μg/100 ml, with three-fourths of the animals having values below 1500 μg/100 ml. A high curvilinear correlation was observed between blood ammonia and plasma free acids in four-fifths of the animals. The data suggest that in addition to ammonia, fatty acids play a significant role in experimental hepatic coma.

0154 DISTRIBUTION AND FATE OF SYNTHETIC LIPID VESICLES IN THE MOUSE: A COMBINED RADIONUCLIDE AND SPIN LABEL STUDY *(E.)* McDougall, I. R.; Dunnick, J. K.; McNamee, M. G; Kriss, J. P. (Stanford Univ. Med. Cent., Calif.). *Proc. Natl. Acad. Sci. USA* 71(9):3487-3491, 1974.

The distribution and fate of single-compartment vesicles, prepared with a spin label (tempocholine chloride) and/or $^{99m}TcO_4$, were studied following i.v. injection into C_3H mice. About 25% of the administered radioactivity was present in the liver 5 min and 30 min after the injection of vesicles composed of phosphatidylcholine and gangliosides, which were sonicated for 5 min (standard preparation). About 10-20% of the radioactivity remained in the circulation. By use of the nonradioactive spin label enclosed within vesicles, intact vesicles were demonstrated in the circulation for 46 min after injection. The distribution of radioactivity from $^{99m}TcO_4^-$ inside vesicles was very different from that of free $^{99m}TcO_4^-$ or of ^{99m}Tc sulfur colloid. Increase in the length of sonication or incorporation of cholesterol into the wall of the vesicles enhanced hepatic levels and reduced blood levels of radioactivity. These same

manipulations also showed the rate of transfer of $^{99m}TcO_4^-$ out of vesicles in dialysis experiments *in vitro*. addition of phosphatidic acid, phosphatidylethanolamine, or phosphatidylserine to the standard constituents did not greatly alter the distribution of radioactivity *in vivo* but did increase the number and type of active coupling sites on the outside of the vesicle. The results indicate that vesicles might be valuable as carriers of diagnostic or therapeutic agents which might be delivered selectively to various target organs or tissues.

0155 STUDIES ON THE SYNTHESIS OF LIVER PHOSPHOLIPIDS AND THE TURNOVER OF PLASMA PHOSPHOLIPIDS IN PREGNANT RATS USING RADIOACTIVE PALMITATE. *(E.)* Schirrmeister, W.; Hummel, L.; Zimmermann, T. (Inst. Pathol. Physiol., Friedrich-Schiller Univ., Jena, Germany). *Acta Biol. Med. Germ.* 32(5):557-560, 1974.

Liver and plasma phospholipids and maternal plasma free fatty acids from 40 pregnant Wistar rats (21 days pregnant) i.v. labeled with ^{14}C-1-palmitate were assayed at various time intervals following injection to obtain the rate of synthesis of liver phospholipid fatty acids and the turnover rate of plasma phospholipid fatty acids. The data were normalized to a constant injected dose of radioactive palmitate (20×10^6 cpm) and a body weight of 300 g. The specific activity of liver phospholipids remained constant from 5-120 min following injection of the label into the mother, indicating that the radioactivity accumulated in the liver phospholipids up to 5 min left at an extremely slow (undetectable) rate. There was virtually no radioactivity left in the plasma free fatty acids 5 min after injection of label and thus no more radioactivity to enter the liver phospholipids. It was postulated that a rapid exchange of label occurred between the individual liver phospholipids. A comparison of the amount of free fatty acids esterified to liver phospholipids (1.02 μM) and the turnover rate of plasma phospholipid fatty acids (0.62 μM/min) indicated that only 60% of the plasma free fatty acids esterified to liver phospholipids was secreted by the liver into the plasma. The remaining 40% was apparently channelled after their hydrolysis into other pathways. The turnover time of plasma phospholipid fatty acids was calculated to be 1.21 hr.

0156 EXPERIMENTS ON THE FORMATION OF $^{14}CO_2$ IN HUMAN LIVER BIOPSIES FROM LABELED FATTY ACIDS WITH DIFFERENT CHAIN LENGTH AND DIFFERENT NUMBER OF DOUBLE BONDS. *(Ger.)* Fischel, P.; Oette, K. (Med. Clin., Univ. Cologne, Germany). *Res. Exp. Med. (Berl.)* 163(1):1-16, 1974.

The formation of $^{14}CO_2$ from albumin bond 1-^{14}C fatty acids in human liver biopsies was studied. The samples were taken from normal subjects and from patients with pancytopenia, myocarditis, diabetes, alcoholism, hepatitis, hyperbilirubinemia, dermatitis herpetiformis, liver cirrhosis, toxoplasmosis, jaundice, hemolytic anemia, Lofgren's syndrome, porphyria cutanea tarda, fatty liver degeneration, and obesity. Regardless of the chain length and number of double bonds, fatty acids were oxidized in proportion to their concentration in the medium. Short-chain and long-chain fatty acids did not influence each other, and glucose only depressed the oxidation of short-chain fatty acids. Ethanol decreased the oxidation of both long-chain and short-chain fatty acids. The molar oxidation rate was essentially determined by the chain length. Similar results were obtained with rat liver slices. A maximum rate of oxidation was found for C_6 fatty acid. The oxidation rate was lowest for long-chain fatty acids. With the exception of stearic acid, they showed only minor differences. Stearic acid, oxidized much more easily, showed ATP formation rates comparable to those of short-chain fatty acids. In view of the small number of patients, the data obtained were not correlated to specific diseases and diets. Both of these factors were, however, of minor importance in comparison with the influence of the type of the fatty acid.

0157 FAILURE OF ADRENALCORTICOSTEROIDS TO INFLUENCE THE MAJOR MECHANISMS OF HEPATIC CHOLESTEROGENESIS. *(E.)* Nervi, F. O.; Dietschy, J. M. (Univ. Texas Southwestern Med. Sch., Dallas). *Biochim. Biophys. Acta* 369(3):351-360, 1974.

Under conditions of carefully controlled dietary intake and cyclic lighting the effect of bilateral adrenalectomy on the alterations in hepatic sterol synthesis caused by diurnal variation in lighting, cholestyramine feeding, fasting i.v. intestinal lipoprotein injection and stress was investigated in female Sprague-Dawley rats. Adrenalectomy caused no alterations in the rate of hepatic CO_2 production or of cholesterol or ketone body synthesis. Cholesterogenesis varied from 255 to 738 nM/g/hr, in the mid-light and mid-dark phases of the light cycle, resp., in control animals; identical variation was found in adrenalectomized animals. Cholestyramine feeding caused a 3-fold increase in hepatic cholesterogenesis in both control and corticosterone-deficient groups. Cholesterol synthesis was markedly suppressed in control rats and in rats with adrenalectomy by fasting for 48 hr and by i.v. injection of intestinal lipoproteins. Finally, stress induced a 10-fold increase in the rate of hepatic cholesterogenesis in both the adrenal-deficient and control animals. These results indicated that adrenalectomized animals responded in an essentially normal manner to all of the major physiological maneuvers tested that are known to affect the rate of hepatic cholesterol synthesis.

0158 REGULATION OF CHOLESTEROL 7α-HYDROXYLATION BY CHOLESTEROL SYNTHESIS IN RAT LIVER. *(E.)* Takeuchi, N.; Ito, M.; Yamamura, Y. (Osaka Univ. Hosp., Japan). *Atherosclerosis* 20(3):481-494, 1974.

Hepatic cholesterol 7α-hydroxylation and cholesterogenesis were determined *in vitro* at various time intervals 6 hr after p.o. or i.v. administration of glucose to fasted rats. Glucose administration to fasted rats enhanced cholesterol 7α-hydroxylation as well as fatty acid or cholesterol synthesis in the

liver, though significant changes were not demonstrated in the cholesterol content of liver and serum throughout the observed periods. Hepatic cholesterol synthesis, as determined by the rate of incorporation of [1-^{14}C]sodium acetate into liver slices, increased 1 hr after oral glucose ingestion (2 ml/100 g of a 50% solution). On the other hand, cholesterol 7α-hydroxylation, also determined by *in vitro* assay using liver slices, was stimulated 2 hr after oral glucose administration, with a lag phase of 1 hr following the induction of cholesterol synthesis. From the results of the experiments on the effect of glucose ingestion it appeared that cholesterol 7α-hydroxylation was correlated with hepatic cholesterogenesis. Hepatic cholesterogenesis was decreased in triparanol-treated rats (8 mg/day p.o for 7 days) and was not stimulated by glucose administration. Triparanol did not affect cholesterol hydroxylation in the fasted state, but did abolish the stimulatory effect of glucose. Short-term feeding of a high-cholesterol (1.5%) diet, which suppressed hepatic cholesterol synthesis, stimulated cholesterol 7α-hydroxylation. Oral administration of glucose to cholesterol-fed rats still further accelerated hydroxylation. In rats with ligated bile ducts, hepatic cholesterogenesis increased 24 hr after bile-duct ligation while hydroxylation decreased 24 hr after the operation but was stimulated after 72 hr. Glucose ingestion by these rats stimulated both cholesterol synthesis and hydroxylation. It is concluded that increased hepatic cholesterogenesis enhances cholesterol 7α-hydroxylase activity; this activity does not depend upon increased fatty acid or other lipid synthesis, altered bile flow or gastrointestinal factors. Increased dietary cholesterol intake also accelerates hydroxylase activity. These mechanisms may preserve homeostasis in the serum cholesterol level, and an altered balance between these two enzyme systems--anabolic and catabolic--probably induces a change in the size of the body cholesterol pool.

0159 DEMONSTRATION AND SOLUBILIZATION OF AN INHIBITOR OF HEPATIC CHOLESTEROL BIOSYNTHESIS FROM RAT LIVER MICROSOMES. *(E.)* Krzemien, J. R.; Haven, G. T. (Coll. Med., Univ. Nebraska, Omaha). *Biochim. Biophys. Acta* 369(1):70-78, 1974.

Acetone powders were prepared from liver microsomes of Sprague-Dawley rats by a modification of the Linn method. The powder cake was washed with ethyl alcohol and dried *in vacuuo* at room temperature for several hr. The solubilized hepatic fraction was suspended in 0.1 M phosphate buffer, pH 7.0, without the addition of any thiol compound. Any hydroxymethylglutaryl-coenzyme A reductase in the preparation was presumably destroyed by exposure to air during ethanol washing and drying at room temperature in the absence of a thiol compound. In a cell-free homogenate system, the buffer-soluble fraction inhibited incorporation of $^{14}C_2$-acetate into cholesterol in a manner similar to that previously reported for the solubilized renal fraction. In a liver slice system, intact hepatic microsomes from fasted animals inhibited cholesterol synthesis from $^{14}C_2$-acetate significantly more than those from fed rats. In contrast, the inhibitory effects of hepatic microsomal acetone powders from fed and fasted rats appeared identical. In intact animals, the solubilized hepatic microsomal fraction caused an apparent 30% inhibition of cholesterol synthesis and a 34% decrease in labeled plasma cholesterol. The results for this system were not statistically significant possibly because of the large intragroup variability. Further purification and characterization of hepatic and renal microsomal factors is required to determine their importance in the physiologic control of hepatic cholesterol synthesis.

0160 EXPERIMENTAL INVESTIGATIONS OF CHOLESTEROL AND CHOLESTEROL ESTER METABOLISM WITH DIFFERENT PRECURSORS IN RAT LIVER AND HUMAN LIVER BIOPSIES AS A FUNCTION OF THE CARBOHYDRATE-FAT CONTENT OF THE DIET. *(Ger.)* Oette, K.; Phlippen, R.; Frotz, H.; Gheorghiu, T.; Lanckohr, H.; Kupfer, I. (Med. Clin., Univ. Cologne, Germany). *Res. Exp. Med. (Berl.)* 163(1):17-30, 1974.

The cholesterol and cholesterol ester metabolism with different precursors were studied in infantile rats and in rat and human liver biopsies as a function of the carbohydrate-fat content of the diet. *In vivo* experiments with rats, using ^{14}C-labeled precursors revealed a great importance of fatty acids as cholesterol precursors in the liver but not in the skeletal muscles, and good oxidizability of stearic acid in the liver. The breakdown products of fatty acids and fructose were preferred precursors of cholesterol in human liver biopsies. The breakdown products of stearic acid were taken up into the cholesterol most intensely. The uptake of fatty acid breakdown products using linoleic acid was not influenced by the fat-carbohydrate content of the diet, but it was proportional to the albumin-bound palmitic acids concentration as demonstrated by rat liver experiments. The high-fat diet caused no increase in the activity of the cholesterol-synthesizing enzyme system. This enzyme system was, however, activated following application of ethanol for 6 days. Considerable changes and variations in the daily diet and in the daily cholesterol uptake caused no changes in the uptake of fatty acid breakdown products in general and of linoleic acid in particular, in cholesterol. The rate of formation of cholesterol esters was low, and the differences between long-chain fatty acids were of minor degree. Small proportions of fatty acids and monosaccharides into cholesterol esters were taken up, and highest absolute uptake was determined for myristic acid. There was no dietary influence on the uptake of ^{14}C-linoleic acid into the cholesterol esters. No preferred synthesis of saturated cholesterol esters was found in human biopsies. The findings indicate little, if any, significance of hepatic cholesterol ester formation for the plasma cholesterol esters.

0161 ORIGIN AND NATURE OF THE PROTEINS OF BILE. II. A COMPARATIVE ANALYSIS OF SERUM, HEPATIC LYMPH AND BILE PROTEINS IN THE DOG. *(E.)* Dive, C.; Nadalini, R. A.; Vaerman, J. P.; Heremans, J. F. (Dep. Intern. Med., Catholic Univ. Louvain, Belgium). *Eur. J. Clin. Invest.* 4(4):241-246, 1974.

The derivation of plasma proteins in bile (except for immunoglobulin A) from the circulation was studied in mongrel dogs. Bile and lymph were collected over a 1/2-hr period, with collection of a blood sample at the mid-time point. The concentrations of individual plasma proteins were determined in the plasma and hepatic lymph by radial immunodiffusion. A biosynthetic labeling method was used in protein excretion studies. After induction of protein deficiency, a donor dog received 75 ml saline containing labeled amino acids (L-tryptophan, L-phenylalanine, L-lysine, L-methionine, and L-leucine). Total radioactivity injected was about 30 mCi. Twelve hr later the donor dog was bled to death and the plasma separated from the collected blood. The radioactive plasma was given to a 2nd dog during a plasmapheresis experiment and 72 hr later, bile, hepatic lymph and blood were collected. For each of the relevant proteins, the specific radioactivity was determined after isolation of the component in the form of a specific immune precipitate. The major plasma proteins in hepatic bile, with the exception of immunoglobulin A, were found to be derived entirely from the plasma. Biliary IgA has two sources, the plasma and local biosynthesis. The transfer of plasma proteins from blood to bile proceeded along 2 parallel pathways, one involving bulk transfer of unmodified plasma, and one based on molecular sieving favoring the transfer of plasma proteins of low molecular weight. The molecular sieve process was completed by the time the plasma proteins reached the interstitial fluid. The sieving membrane was therefore assigned anatomically to the vascular walls, which have the properties of an isoporous membrane with a pore radius of 102 Å. No further molecular sieving took place during the crossing of the second barrier between blood and bile, that is, the epithelial sheet separating interstitial fluid from the bile.

0162 THE BINDING OF NATURAL AND SYNTHETIC DYES TO BOUND PROTEINS OF THE LIVER. *(Cs.)* Jirsa, M.; Hykes, P.; Schinkmannova, L. (Fac. Gen. Med., Charles Univ., Prague, Czechoslovakia). *Cas. Lek. Cesk.* 112(45):1398-1401, 1973.

Quantities of various dye anions bound *in vitro* to proteins Y and Z in liver cytosol were determined spectrophotometrically. Mixtures of 0.01 ml of 0.06 M dye solutions and 3 ml of liver cytosol obtained from homogenized, centrifuged Wistar rat liver were filtered at least twice through a gel column and the quantity of bound dye was determined by measuring E at 278 nm and E at the absorption maximum for each dye. Depending upon their binding to proteins Y and Z, dyes can be divided into 3 groups. Group (1) consists of dyes that are bound primarily to protein Y and are excreted by the liver. This group includes BSP, phenol tetrachlorophthalein, protoporphyrin, conjugated bilirubin, and taurobilirubin. Group (2) consists of dyes which are bound to about the same extent to proteins Y and Z or to a greater extent by Z. These include iodinated BSP, bengal rose, and bilirubin. Group (3) consists of dyes which are not bound to either protein and includes phenol red. From Kav values the molecular weight of protein Y was calculated to be 37,000 and that of protein Z, 17,000. The failure of phenol red to bind to either Y or Z is attributed to its polar $-SO_3$ group which retards binding, although a $-SO_3$ group on the phenol nuclei has no appreciable effect. Uroporphyrin, which has 8 carboxyl groups, and protoporphyrin, which has 2 carboxyl groups, are bound in the same way. However, the very polar uroporphyrin is excreted in the urine. This is apparently due to the inability of uroporphyrin to cross the bile pole of the liver cell: it diffuses back into the blood and is excreted by the kidneys. It is surprising that some dyes, e.g. iodinated BSP and bengal rose, are bound to a greater extent by protein Z, which is less typical for liver cells, and are also found in other organs, but are still excreted in large quanitities by the liver.

0163 EVIDENCE FOR THE BIOSYNTHETIC DIFFERENCE BETWEEN ISOLATED MITOCHONDRIA AND MICROSOMES FROM GUINEA-PIG AND RAT LIVER REGARDING LYSOPHOSPHATIDIC ACID, PHOSPHATIDIC ACID, CDP-DIGLYCERIDE, PHOSPHATIDYLGLYCEROL, AND CARDIOLIPIN. *(E.)* Davison, J. B.; Stanacev, N. Z. (Fac. Med., Univ. Toronto, Canada). *Can. J. Biochem.* 52(10):936-939, 1974.

The activities of marker and synthetic enzymes were measured in isolated mitochondria and microsomes from guinea pig and rat liver. Of the 2 marker enzymes studied, the activity of NADPH-cytochrome *c* reductase was higher in guinea pig than in rat liver microsomes, whereas the activity of glucose-6-phosphatase was almost the same in both species. The microsomal content in the mitochondria determined by the 2nd marker enzyme was twice that determined by the 1st. The activity of the synthetic enzyme acyl-coenzyme A (CoA):glycero-3-phosphatase acyltransferase was predominantly in microsomes and was 3 times higher in guinea pig microsomes than in rat liver microsomes. The activity of CDP-diglyceride-glycero-3-phosphate phosphatidyltransferase was much higher in rat liver than in guinea pig mitochondria; it was negligible in microsomes of both species. Activity of CTP:phosphatidic acid cytidyltransferase was predominantly in microsomes in both species. In guinea pig mitochondria, but not in rat liver mitochondria, the activity of acyl-CoA:glycero-3-phosphate acyltransferase paralleled the activity of microsomal marker enzymes in this fraction. In both species activity of mitochondrial CTP:phosphatidic acid cytidyltransferase paralleled the activity of the microsomal marker enzymes in mitochondria. Formation of lysophosphatidic acid is an intrinsic mitochondrial process in rat liver but can be attributed to microsomes in the guinea pig liver. In both guinea pig and rat liver mitochondria, formation of CDP-diglyceride can be attributed to microsomal content, while the formation of phosphatidylglycerophosphate, phosphatidylglycerol, and cariolipin is an intrinsic mitochondrial process.

0164 ORNITHINE DECARBOXYLASE ACTIVITY, NUCLEIC ACIDS AND CELL TURNOVER IN THE LIVERS OF PREGNANT RATS. *(E.)* Campbell, R. M.; Fell, B. F.; Mackie, W. S. (Rowett Res. Inst., Bucksburn, Scotland). *J. Physiol. (Lond.)* 241(3):699-713, 1974.

Nucleic acid content, cell turnover, and ornithine decarboxylase activity were studied in livers of 28 pregnant and 24 nonpregnant rats. All animals received 20 g food/day. Liver weight, as well as total hepatic protein, and RNA and DNA content, increased markedly during the 2nd half of pregnancy when compared to controls. Total RNA concentration increased from the 15th day of gestation. Between the 12th and 18th day of pregnancy liver weight, total RNA, total DNA and mean liver cell nuclear volume increased in parallel at approximately the same rate of 6-10%/day. Ornithine decarboxylase activity, determined by the rate of liberation of $^{14}CO_2$ *in vitro* from carboxyl-labeled ornithine, was enhanced in pregnancy by the 5th day of gestation and rose to peak activity at the 18th day. The mitotic index of hepatic parenchymal cells was elevated in the first and last stages of pregnancy. The later peak in mitosis was associated with a rapid fall in mean nuclear volume. There was histological evidence of cell turnover in the liver of pregnant rats; enlargement of the liver was associated with both hyperplasia and cell deletion. It is concluded that in rats, pregnancy is associated with liver cell enlargement and proliferation, and also with parenchymal cell deletion.

0165 COBALT INDUCTION OF HEPATIC HEME OXYGENASE; WITH EVIDENCE THAT CYTOCHROME P-450 IS NOT ESSENTIAL FOR THIS ENZYME ACTIVITY. *(E.)* Maines, M. D.; Kappas, A. (Rockefeller Univ., New York, N.Y.). *Proc. Natl. Acad. Sci. USA* 71(11):4293-4297, 1974.

The effects of cobalt on heme catabolism were studied in male Sprague-Dawley rats. Animals were injected with cobalt chloride (60 mg/kg, s.c.), with methemalbumin (40 μM/kg, i.p.), or with both cobalt chloride and methemalbumin twice at 24-hr intervals. Liver microsomal heme oxygenase and mixed-function oxidase activity were subsequently assayed. Cobalt chloride stimulated hepatic microsomal heme oxidation to levels up to 800% those of saline-injected controls and also caused a 20% decrease in both liver weight and total microsomal protein. In contrast, marked decreases were produced in microsomal oxidation of ethylmorphine (80%), and in cytochrome P-450 (60-70%) and heme (30-50%) contents. Cobalt chloride treatment did not affect heme oxidation by the spleen heme oxygenase system. The rate of heme oxidation by hepatic microsomal enzymes and the microsomal content of cytochrome P-450 were found to be unrelated. This conclusion was reached from studies in which microsomal heme oxygenase activity from cobalt-treated animals could be increased by 900% above control levels in the same microsomal preparation in which cytochrome P-450 content was decreased to spectrally unmeasurable amounts after incubation with 4M urea. The same treatment eliminated ethylmorphine demethylation and decreased microsomal NADPH-cytochrome c reductase (EC 1.6.2.4) activity by 75%. It was concluded: 1) that the hepatic microsomal enzyme system that oxidizes heme compounds is not the same as that which metabolizes drugs; 2) cytochrome P-450 is not essential for the oxidation of heme by liver cells; 3) there is no direct relation between the rate of heme oxidation and the level of NADPH-cytochrome c reductase activity; and 4) the oxidation of heme is protein-dependent and the active proteins are inducible, but are different from those involved in drug metabolism.

0166 EFFECT OF INHIBITORS OF PROTEIN AND RIBONUCLEIC ACID SYNTHESIS ON THE ALTERATION IN BILIARY BILIRUBIN EXCRETION AND NON-ERYTHROPOIETICALLY DERIVED BILIRUBIN SYNTHESIS IN RATS AFTER α-NAPHTHYLISOTHIOCYANATE ADMINISTRATION. *(E.)* Triager, G. J.; De Repentigny, L.; Plaa, G. L. (Dep. Pharmacol., Univ. Montreal, Canada). *Biochem. Pharmacol.* 23(20): 2845-2856, 1974.

In an attempt to explain the inhibition of α-naphthylisothiocyanate (ANIT)-induced hyperbilirubinemia by inhibitors of protein and RNA synthesis, the effect of cycloheximide, ethionine and actinomycin D on the increased bilirubin formation produced by ANIT (150 mg/kg by gastric lavage) was studied in male Sprague-Dawley rats. Cycloheximide (2 mg/kg i.p.) and ethionine (400 mg/kg i.p.) administered 1 and 2 hr prior to ANIT treatment resp., inhibited the ANIT-induced increase in biliary bilirubin excretion and incorporation of δ-aminolevulinic acid-^{14}C into bilirubin. Cycloheximide was found to be more effective than ethionine in diminishing the stimulation of bilirubin production by ANIT. Pretreatment with actinomycin D (100 μg/kg i.p.) 1 hr prior to ANIT prevented the ANIT-induced increase in biliary bilirubin excretion and produced a decreased but irregular incorporation of δ-aminolevulinic acid-^{14}C into bilirubin. β-Naphthylisothiocyanate (300 mg/kg by gastric lavage), which does not produce hyperbilirubinemia, failed to enhance biliary bilirubin excretion. The results suggest that the enhancement of bilirubin formation by ANIT plays a role in the overall hyperbilirubinemic response seen after ANIT administration. The inhibition of the enhanced bilirubin formation by cycloheximide and ethionine may partially account for their inhibition of ANIT hyperbilirubinemia.

0167 POSTNATAL DEVELOPMENT CHANGES IN HEPATIC BILIRUBIN UDP-GLUCURONYL TRANSFERASE: STUDIES ON THE SOLUBILIZED ENZYME. *(E.)* Krasner, J.; Juchau, M. R.; Yaffe, S. J. (State Univ. New York Sch. Med., Buffalo). *Biol. Neonate* 23(5/6):381-390, 1973.

In vitro studies were conducted to compare the kinetic parameters of solubilized and unsolubilized mouse hepatic microsomal bilirubin UDP-glucuronyl transferase during varying stages of postnatal development. Activity of bilirubin glucuronyl transferase in mouse liver homogenates was low at birth, and reached a peak at 14 days of age, 2-3 times greater than adult values. Kinetic studies showed marked differences between microsomes from adult and 14-day-old animals. Michaelis-Menten constants (K_m) were 5.9×10^{-5} M and 4×10^{-4} M resp. at these two ages. K_m values increased from 5.9×10^{-5} M to 6.7×10^{-4} M when enzymes derived from adult animals were solubilized with deoxycholate. In contrast, solubilization did not modify the K_m values in preparations from 14-day-old mice. The newborn K_m value for unsolubilized enzyme was 8.8×10^{-5} M and 6.7×10^{-4} M for the

solubilized preparation. Pretreatment of pregnant animals with barbiturates to induce enzymic activity was necessary for the kinetic studies in the newborn. These findings indicate that different forms of the enzyme exist at the several stages of development studied. Alterations produced by solubilization procedures suggest a common structural form which undergoes alteration during maturation.

0168 THE VIABILITY OF PARENCHYMAL LIVER CELLS ISOLATED FROM YOUNG AND OLD RATS. *(E.)* Van Bezooijen, C. F. A.; Van Noord, M. J.; Knook, D. L. (Inst. Exp. Gerontol., Rijswijk, Netherlands). *Mechanisms Ageing Dev.* 3(2):107-119, 1974.

Isolated parenchymal cells from young and old rats were utilized in *in vitro* studies on cellular aging processes in the rat liver, and the various criteria for the viability of these cells discussed. Under ether anesthesia, the liver and portal vein of female WAG/Rij rats (2 week - 36 month) were dissected free and left *in situ*. A cannula was inserted into the portal vein. After a rapid (25 ml/min) infusion of about 15 ml of dissociation medium (standard medium plus 0.05% collagenase and 0.10% hyaluronidase) the liver blanched and then was perfused *in situ* with 50 ml dissociation medium at 37 C at a rate of 3 ml/min. The liver was excised and the isolated parenchymal cells assayed for viability. Trypan blue exclusion demonstrated the impermeability of the plasma membrane. The ultrastructural integrity of the impermeable cells was confirmed by electron microscopic observations. Oxygen consumption as measured by a Clark oxygen electrode was comparable to that of hepatic parenchymal cells in intact tissue. Succinate or other tricarboxylic acid cycle intermediates did not increase the respiration of isolated cells, but a marked stimulation was observed if cell debris was present. The endogenous respiration/million cells was higher for cells from old rats than for cells from young rats. This increase could be attributed to an increase in average cell size resulting from a shift to polyploid cells with age. The results obtained show that the cells isolated by this method are comparable to intact liver tissue with respect to endogenous respiration, membrane permeability, and ultrastructural appearance.

0169 CITRULLINE SYNTHESIS IN RAT TISSUES AND LIVER CONTENT OF CARBAMOYL PHOSPHATE AND ORNITHINE. *(E.)* Raijman, L. (Univ. South. California Sch. Med., Los Angeles). *Biochem. J.* 138(2): 225-232, 1974.

The intracellular localization and activity of ornithine carbamoyltransferase were studied in whole liver homogenates and hepatocyte mitochondrial preparations from male Wistar rats. Rat liver ornithine carbamoyltransferase appeared to be located exclusively in the mitochondria. The activity that was found in the soluble fraction was indistinguishable from mitochondrial ornithine carbamoyltransferase by simple kinetic criteria, and seemed to result from breakage of mitochondria during homogenization. Of several rat tissues studied, only the liver and small intestinal mucosa contained significant amounts of ornithine carbamoyltransferase. The activity in intestinal mucosa was less than 1/1000th of that in liver. The distribution coincides qualitatively with that of carbamoyl phosphate synthetase I and its cofactor, acetylglutamate. The rat liver contents of carbamoyl phosphate and ornithine were 0.1 and 0.15 μM/g wet weight, resp. On the basis of these values, it is proposed that the ornithine carbamoyltransferase activity of liver *in vivo* may be much lower than its maximal activity *in vitro* might suggest.

0170 HYDROXYLATION OF TAUROLITHOCHOLATE BY ISOLATED HUMAN LIVER MICROSOMES. II. CYTOCHROME P-450 DEPENDENCY. *(E.)* Czygan, P.; Greim, H.; Trulzsch, D.; Rudick, J.; Hutterer, F.; Schaffner, F.; Popper, H.; Rosenthal, O.; Cooper, D. Y. (Mt. Sinai Sch. Med., City Univ. New York, N.Y.). *Biochim. Biophys. Acta* 354(2):168-171, 1974.

The dependency of conversion of taurolithocholate to taurohyodeoxycholate by 6α-hyroxylation on cytochrome P-450 was studied in isolated normal human liver microsomal preparations in the presence of NADPH. The reaction rate in the presence of different concentrations of carbon monoxide (CO) showed a dose-dependent inhibition with 50% inhibition occurring at a partition constant of 1.8. Inhibition of 6α-hydroxylation by CO *in vitro* was maximally reversed with exposure to monochromatic light at 450 nm with a smaller reversal in 1 experiment occurring with exposure to light at 419 nm. These findings indicated for the first time that the human liver microsomal 6α-hydroxylating system is cytochrome P-450 dependent.

0171 IMMUNOLOGICAL AND ELECTRON MICROSCOPIC FINDINGS FOLLOWING HEPATIC AUTOANTIBODY FORMATION. *(Ger.)* Intorp, H. W.; Stohlmann, J.; Gruber, H.; v. Bassewitz, D. B. (Univ. Med. Polyclin., Munster, Germany). *Verh. Dtsch. Ges. Inn. Med.* 79:654-657, 1973.

Rabbits were administered 6 to 14 injections of human or rabbit liver tissue preparations at weekly intervals, after which the antisera obtained were investigated by complement binding test, passive hemagglutination test, and double diffusion test in agargel. Tissue samples were also investigated under electron microscopy. Immunization with human liver preparations caused antibody formation against numerous soluble and insoluble hepatic antigens, while liver-specific reactions were not detected with non-absorbed antisera. Following absorption, the antisera reacted only with human liver preparations; reactions with kidney and spleen preparations were also detected occasionally. The liver-specific antigens of the soluble tissue fraction were also detected in monkeys. Immunization with rabbit liver homogenates resulted in the formation of antibodies against 2 soluble antigens localized in the hepatic and renal mitochondria in rabbits immunized with 4 injections, and in the formation of antibodies against 2 liver-specific antigens in rabbits immu-

nized with 8 or more injections. Liver biopsies taken shortly before the animals were sacrificed revealed broadening of the periportal fields, proliferation of collagen fibers, lymphocyte and plasma cell infiltration, and proliferation of Kupffer's astrocytes containing phagocytized material with paracrystalline substructure. The findings indicate hepatic autoantibody formation following immunization with heterologous and homologous liver tissue, and cellular immune reactions are confined primarily to the bile ducts and liver parenchyma.

0172 EFFECT OF SERA OBTAINED FROM NORMAL AND PARTIALLY HEPATECTOMIZED RATS AND PATIENTS ON THE GROWTH OF CELLS IN TISSUE CULTURE. *(E.)* Demetriou, A. A.; Seifter, E.; Levenson, S. M. (Albert Einstein Coll. Med., Bronx, N. Y.). *Surgery* 76(5):779-785, 1974.

The effect of posthepatectomy and normal rat or human serum on cultured E_1 cells derived from rat hepatocytes was studied. Hepatectomies (70%), sham hepatectomies, and unilateral nephrectomies were performed in rats, and filtered, dialyzed serum obtained 24 hr later was added to the culture medium. Addition of serum (5 and 10%) from hepatectomized rats caused considerable stimulation of cell growth; this effect was not altered when serum from rats which underwent hepatectomy and unilateral nephrectomy was added to the medium. Serum from normal, sham-hepatectomized or unilaterally nephrectomized rats produced no effect on cell growth. Similarly, serum from normal human beings or from a patient who had undergone a negative laparotomy following trauma produced no effect. In contrast, serum (5 and 10%) from a man 14 days following 75% hepatectomy done because of liver injury stimulated cell growth considerably. Initial attempts to isolate the stimulating factor revealed that the active compound(s) is entirely filterable through cellophane membrane and has molecular weight of less than 12,000. It is concluded that: (1) normal serum does not contain an inhibitor of cell growth; (2) a factor stimulating cell growth *in vitro* is present in the serum of rats and man following partial hepatectomy; and (3) this stimulating factor has a molecular weight of less than 12,000.

0173 HEPATIC VITAMIN B_{12} RELEASE AND TRANSCOBALAMIN II SYNTHESIS IN THE RAT. *(E.)* Cooksley, W. G. E.; England, J. M.; Louis, L.; Down, M. C.; Tavill, A. S. (Med. Res. Comm. Clin. Res. Cent., Harrow, England). *Clin. Sci. Mol. Med.* 47(6):531-545, 1974.

The release of ^{57}Co-labeled vitamin B_{12} and the synthesis of transcobalamin II by isolated perfused rat liver were studied 10-42 days after i.m. administrations of 3 μCi (approximately 20 ng) of radioactive cyanocobalamin to 3-4-month-old male Sprague-Dawley rats. The rate of release of [^{57}Co] vitamin B_{12} into plasma and bile was linear and constituted approximately 0.9% and 0.3%, resp., of the initial hepatic radioactivity/hr of perfusion. [^{57}Co]B_{12} released into the plasma was bound to transcobalamin II. Saturation of the total transcobalamin II by the addition of 40 ng/ml of cyanocobalamin to homologous rat plasma (incubated at 37 C for 10 min) before perfusion resulted in the appearance of the hepatic [^{57}Co]B_{12} in the free form. These findings were compatible with *in vivo* observations of urinary and biliary excretion rates of [^{57}Co]B_{12}. Rats previously injected 3 μCi i.m. of [^{57}Co]B_{12} were injected i.m. with unlabeled cyanocobalamin in doses of 300 pM, 3.0 nM, 30 nM, or 300 nM in 2 divided doses at 2 hr intervals and released between 1.55% to 7.5% of the final hepatic radioactivity as urinary [^{57}Co]B_{12}. Pairs of rats injected i.m. with 300 pM, 600 pM, 1.2 nM, 3.0 nM, 30 nM, and 300 nM of cyanocobalamin (each dose containing 0.12 μCi of cobalt-labeled cyanocobalamin) showed a sharp increase in urinary recovery of injected B_{12} for doses between 600 pM and 3.0 nM, with a gradual rise to an 80% recovery in 4 hr at the maximum dose. Biliary excretion of [^{57}Co]B_{12} in 2 rats was linear at a rate of 0.19%/hr and 0.22%/hr of the final hepatic radioactivity. Liver damage produced by hypoxemia was associated with a fall in the rate of [^{57}Co]B_{12} release. Transcobalamin II release occurred at a linear rate of almost 20 times that required for the binding of newly released hepatic vitamin B_{12}. Cycloheximide at a dose sufficient to inhibit release of transcobalamin II (perfusate plasma concentration of 10-18 μM/liter) did not prevent the release of [^{57}Co]B_{12} from the liver into plasma or bile. Alteration of the perfusate composition in terms of the relative amounts of vitamin B_{12} plasma concentrations or unsaturated transcobalamin II concentrations had no effect on the rate of [^{57}Co]B_{12} release into plasma or bile. The fluxes of hepatic vitamin B_{12} and transcobalamin II appear to be very rapid, and the release of vitamin B_{12} by the rat liver appears to be controlled in the short term by factors other than transcobalamin II synthesis and the concentration of vitamin B_{12} or unsaturated transcobalamin in the plasma.

0174 REGULATION OF THE MITOCHONDRIAL OXIDATION-REDUCTION STATE IN INTACT HEPATOCYTES BY CALCIUM IONS. *(E.)* Otto, D. A.; Ontko, J. A. (Univ. Oklahoma Med. Res. Found., Oklahoma City). *Biochem. Biophys. Res. Commun.* 61(2):693-700, 1974.

The regulatory influence of calcium on mitochondrial NADH:NAD was investigated in intact hepatocytes from male Holtzman rats. Addition of 1.25 mM calcium chloride to incubated liver cells increased the β-hydroxybutyrate:acetoacetate production ratio from 0.32 to 1.72. A half-maximal production ratio increase occurred at a $CaCl_2$ concentration of 100 μM, with a maximum ratio of 2.0 occurring at 1 mM $CaCl_2$ after steady state (20 min) was achieved. Higher calcium levels did not cause reversal. Chloride salts of barium, cadmium, copper, manganese, strontium, zinc, aluminum, cerium, and lanthanum exerted no such effect, indicating the high specificity of the calcium-induced NADH:NAD elevation. One mM $CaCl_2$ increased the β-hydroxybutyrate:acetoacetate ratio in hepatocytes incubated without added substrates (0.02 to 0.07). The ratio was also increased in the presence of: 10 mM fumarate (0.20 to 0.27); 10 mM L-malate (0.19 to 0.34); 10 mM

L-glutamate (0.10 to 0.22); 10 mM α-ketoglutarate (0.19 to 0.30); 20 mM β-hydroxybutyrate (1.51 to 3.73); and 10 mM L-lactate (0.36 to 0.62). Calcium decreased the β-hydroxybutyrate:acetoacetate ratio from 1.07 to 0.80 when cells were incubated with 10 mM succinate. Calcium did not alter the lactate:pyruvate ratio. The calcium-induced NADH:NAD alteration appeared to be mediated by direct interaction of mitochondrial calcium and not *via* primary effects on the NADH:NAD ratio in the cytosol. The calcium-induced NADH:NAD elevation was reversed by the uncoupler 1799. Calcium appeared to interact with 1 or more respiratory carriers of the inner mitochondrial membrane, with the elevation of NADH:NAD ratio possibly due to the restrictive effect of calcium on NADH utilization.

0175 PORTAL VEIN OCCLUSION IN THE RAT: A NEW TECHNIQUE. *(E.)* Demetriou, A. A.; Seifter, E.; Levenson, S. M. (Albert Einstein Coll. Med., Bronx, N.Y.). *J. Surg. Res.* 17(4):253-254, 1974.

A new technique is described for producing gradual total portal vein occlusion in rats. In this one-step procedure, a dry Ameroid constrictor is placed around the portal vein. As the Ameroid ring swells, the portal vein is slowly occluded. Using this experimental technique, the portal vein was successfully occluded in 20 rats within 7-10 days with no mortalities. None of the rats developed ascites; however, splenic size increased 2-fold. All animals showed extensive inflammatory reaction around the constrictor with development of adhesions.

0176 THE EFFECT OF VASOPRESSIN ON PORTAL HYPERTENSION FOLLOWING HEPATECTOMY. *(E.)* Sirinek, K. R.; Thomford, N. R. (Ohio State Univ. Coll. Med., Columbus). *Surg. Gynecol. Obstet.* 139 (4):573-577, 1974.

In order to study the problem of portal hypertension and splanchnic sequestration which occurs following major hepatic resection, the pharmacologic control of portal hypertension following hepatectomy has been evaluated in dogs. The marked increase in portal vein pressure associated with major hepatic resection was confirmed in control experiments. Administration of vasopressin (0.4 U/ml, 50 ml/hr i.v.) in advance of hepatectomy prevented portal hypertension following hepatectomy but caused a 30% reduction in cardiac output. However, when some vasopressin-treated dogs were given isoproterenol (0.004 mg/ml, 25 ml/hr i.v.) at the time of hepatectomy, the vasopressin-induced reduction in cardiac output was corrected. The addition of isoproterenol did not interfere with the prevention of portal hypertension by vasopressin. Results of these studies suggest that the administration of vasopressin alone or in combination with isoproterenol to selected patients undergoing major hepatic resection may prevent the life-threatening sequestration of blood in the splanchnic vascular bed.

0177 USE OF SELECTIVE, INTRAHEPATIC, PORTAL VENOGRAPHY AND IN VIVO COLORATION IN PLANNING SEGMENTAL HEPATIC RESECTION. *(E.)* Gross, G.; Goldberg, H. I.; Schrock, T. R. (Univ. California Sch. Med., San Francisco). *Am. J. Roentgenol. Radium Ther. Nucl. Med.* 122(2):327-332, 1974.

Selective intrahepatic portal venography and coloration were performed in 5 dogs and 1 newborn lamb. Toluidine blue was injected into hepatic segments *via* a branch of the portal vein so as to visualize the boundary of the segment. The vascular supply and drainage, as well as surface boundaries, were easily identified by the venogram and coloration, permitting successful segmental resection. Portal venography and instillation of the dye did not result in any histologic changes or change in liver function or portal pressure. The technique of selective intrahepatic portal venography and segmental coloration by way of umbilical vein catheterization may thus be useful in facilitating segmental hepatic resection in humans.

0178 COMPARISON OF THE EFFECTS OF TWO-THIRDS HEPATECTOMY AND BILE DUCT LIGATION ON HEPATIC EXCRETORY FUNCTION. *(E.)* Klaassen, C. D. (Univ. Kansas Med. Cent., Kansas City). *J. Pharmacol. Exp. Ther.* 191(1):25-31, 1974.

The plasma disappearance and biliary excretion of sulfobromophthalein (100 mg/kg i.v.), indocyanine green (10 mg/kg i.v.), procainamide ethobromide (10 mg/kg i.v.), and ouabain (4 mg/kg i.v.) were compared in various groups of control, bile duct-ligated, and 2/3 hepatectomized male Sprague-Dawley rats. Although the hepatectomized rats had livers weighing only 40-45% of the controls, they excreted the 4 drugs into the bile at a rate of 60-65% of controls and produced bile at a rate of 80-90% of controls. Both bile duct-ligated and hepatectomized rats showed decreased rates of disappearance for all 4 drugs from the plasma, with BSP and indocyanine green disappearance being influenced to a greater extent by bile duct ligation and procainamide ethobromide and ouabain being more affected by hepatectomy. Plasma BSP at 30-min for hepatectomized and bile duct-ligated rats were 0.4 and 0.6 mg/ml as compared to 0.2 mg/ml for controls (6-8 rats per group). Plasma indocyanine green levels at 30-min for hepatectomized and bile duct-ligated rats were 20 and 40 μg/ml, resp., as compared to 10 μg/ml for controls (6-8 rats per group). Plasma procainamide ethobromide values at 30-min for hepatectomized and bile duct-ligated rats were about 2.5 and 1.8 μg/ml as compared to a little over 1 μg/ml for controls (9-12 rats per group). Plasma ouabain levels at 30-min for hepatectomized and bile duct-ligated rats were about 1.8 and 2.0 μg/ml as compared to a control value of less than 1 μg/ml (6-9 rats per group). The results suggest that normal plasma disappearance of all 4 drugs is dependent both on hepatic mass and transport from liver to bile. It was suggested that hepatic mass is more important for the clearance of procainamide ethobromide and ouabain and that bile transport is more important for the clearance of BSP and indocyanine green.

0179 BILE SALT METABOLISM AND BILE FORMATION IN THE RAT WITH A PORTACAVAL SHUNT. *(E.)* Herz, R.; Paumgartner, G.; Preisig, R. (Dep. Clin. Pharmacol., Univ. Bern, Switzerland). *Eur. J. Clin. Invest.* 4(4):223-228, 1974.

Biliary secretion of bile salts and bile flow were studied in male Sprague-Dawley rats with liver atrophy resulting from end-to-side portacaval shunts and in sham-operated controls. Three weeks after shunt operation the liver to body weight ratio was reduced by 41%. Bile flow and bile salt excretion were assessed in 13 shunted and 15 sham-operated animals. Bile was collected for a single 10 min period starting 5 min after cannulation. In 15 shunted and 14 sham-operated rats a single 10 min bile sample was collected; the animals were then sacrificed and the liver and intestine removed. Total bile salt pool was obtained as the sum of total bile salts measured in the small and large intestine, in the liver and in the bile. Cholate pool size was calculated from the total bile salt pool size and the percentage of cholic acid in the bile salt mixture of bile; cholic acid half-life and fractional turnover were determined. Hepatic atrophy was accompanied by reduced bile flow (4.9 and 5.9 µl/min/100 g body weight in shunted and sham-operated animals, resp. Bile salt pool size and bile salt secretion were not significantly altered by portacaval shunt. Shunted animals maintained a normal bile salt pool size by increasing bile salt synthesis/U liver weight. Cholate synthesis, calculated from cholate pool size and ^{14}C-cholate turnover rate, was 3.3 and 1.8 µM/24 hr/g liver in shunted and sham-operated rats, resp. It is concluded that the atrophic liver in the rat with a portacaval shunt is able to maintian normal bile salt secretion rates and that the observed decrease of bile flow results from a diminution of bile-salt-independent bile formation.

0180 SECRETION OF SOME HEAVY METALS IN THE BILE OF THE RAT. *(Cz.)* Tichy, M.; Cikrt, M. (Inst. Hyg. Epidemiol., Prague, Czechoslovakia). *Pracov. Lek.* 24(5):187-189, 1972.

Aqueous solutions of $^{64}CuCl_2$, $^{52}MnCl_2$, $^{203}HgCl_2$, and $^{210}Pb(NO_3)_2$ were injected i.v. in quantities far below toxic doses into rats with bile fistulas, and the secretion of these heavy metals was measured in the bile. In 24-hr bile samples 31.1% of the administered Cu^{++}, 26.7% of the Mn^{++}, 3.8% of the Hg^{++}, and 6.7% of the Pb^{++} were secreted. Electrophoresis of the bile on polyacrylamide disks showed that distribution of Cu^{++} and Mn^{++} in the various bile fractions was about the same, but their distribution differed from that of Pb^{++} and Hg^{++}. Determination of total Mn by atomic absorption spectroscopy and bound Mn by electron paramagnetic resonance (EPR) spectra demonstrated that the quantity of Mn present in complexes varies greatly and depends upon the way the bile has been stored (temperature, exposure to light) and the pH of the bile. The quantity of free Mn^{++} was increased by acidification, reduction with sodium hydrosulfite, and ashing of the bile. There intensity of the EPR signal was reduced by 25-40% by addition of a $MnCl_2$ solution to normal rat bile, by 70% when $MnCl_2$ was added to acidified rat bile, and by 30-40% when the bile was boiled for a short time. These findings indicate that most of the Mn is present in complexes with low molecular weight compounds. A comparison of stability constants of complexes formed by the 4 heavy metals studied with cystein, glycine, disodium ethylenediaminetetraacetate, and sulfides showed that Mn^{++} formed the least stable complexes, while Hg^{++} formed very stable complexes, especially with compounds containing sulfhydryl groups. It is hypothesized that Mn^{++} is secreted as a cation after diffusing through the wall between the blood stream and the bile ducts or through the hepatocyte. Mn^{++} then forms complexes in the bile. In the hepatocyte heavy metal cations can form complexes with low molecular weight compounds (bile pigments, bile acids) with which they are secreted in the bile.

0181 PRE-TREATMENT INFLUENCES ON SGPT LEVELS AND LIVER DAMAGE AFTER INHALATION OF CCl_4. *(E.)* Magos, L.; Butler, W. H.; White, I. N. H.; Green, A. (Med. Res. Counc. Lab., Carshalton, England). *Life Sci.* 15(9):1631-1637, 1974.

Ten groups of male albino rats (4 or 8 animals/group) were subjected to different schedules of phenobarbitone treatment and/or starvation for the 48-hr period preceding a 4 hr exposure to 1150 ppm CCl_4 in a dynamic inhalation chamber. Phenobarbitone sodium was given i.p. 24 hr or 48 hr before exposure in a dose of 80 mg/kg followed by a second dose of 50 mg/kg 6 hr later. In starvation, food was withdrawn either from 48-24, 48-0, or 24-0 hrs before exposure. Some animals were killed after the 48 hr pre-exposure period and liver cytochrome P-450 determined. By these treatments a gradual increase in liver cytochrome P-450 was achieved prior to exposure with a 4-fold difference between the lowest, in rats starved 24 hr before exposure, and the highest, in rats given the first phenobarbitone injection 48 hr before exposure. SGPT activity in control rats was 26.5 and this was not affected by starvation or phenobarbitone treatment or their combination. Rats were killed 20 hr after exposure. Neither SGPT activity nor microscopic liver damage correlated with the pre-exposure values of P-450. It is suggested that factors other than the level of cytochrome P-450 determine SGPT levels and liver damage 20 hr after exposure to CCl_4.

0182 BIOCHEMICAL MECHANISM OF HEPATIC NECROSIS INDUCED BY AROMATIC HYDROCARBONS. *(E.)* Reid, W. D.; Krishna, G.; Gillette, J. R.; Brodie, B. B. (Natl. Heart Lung Inst., Bethesda, Md.). *Pharmacology* 10(4):193-214, 1973.

The biochemical mechanism of aromatic hydrocarbon-induced liver necrosis was studied in male Sprague-Dawley rats and male C57 Black/6 mice which received a single i.p. injection of various concentrations of ^{14}C-labeled bromobenzene or other halogenated hydrocarbons. Animals developed centrilobular hepatic necrosis within 24 hr after administration of bromobenzene (4.85 mM/kg). SKF 525-A or piperonyl butoxide injected concurrently with bromobenzene completely

blocked necrosis indicating that the hydrocarbon must be metabolized before it can exert its effects. Animals pretreated with phenobarbital (80 mg/kg i.p. daily x 3) to induce hepatic microsomal cytocyrome P-450 enzymes showed increased susceptibility to the damaging effects of bromobenzene (1.45 mM/kg) and other halogenated hydrocarbons. The hepatic necrosis was preceded by covalent binding of substantial amounts of ^{14}C-bromobenzene to liver proteins. Autoradiographs of livers from animals previously injected with ^{14}C-bromobenzene showed that most of the covalently bound material was localized within the necrotic centrilobular hepatocytes strongly suggesting a causal relationship. The livers of mice pretreated with diethyl maleate to deplete hepatic glutathione stores were more sensitive to the destructive effects of bromobenzene than were controls. These results imply that covalent binding of toxic metabolites may be an important mechanism in the pathogenesis of tissue lesions induced by many substances.

0183 THE METABOLISM OF (-)-OCTANOYLCARNITINE IN PERFUSED LIVERS FROM FED AND FASTED RATS: EVIDENCE FOR A POSSIBLE REGULATORY ROLE OF CARNITINE ACYLTRANSFERASE IN THE CONTROL OF KETOGENESIS. *(E.)* McGarry, D.; Foster, D. W. (Univ. Texas Southwestern Med. Sch., Dallas). *J. Biol. Chem.* 249(24):7984-7990, 1974.

The role of hepatic carnitine acyltransferase in the regulation of ketone body formation was studied in isolated perfused livers from male Sprague-Dawley rats which were starved for 24 hr. In agreement with previous findings, perfused livers from the fasted rats converted oleic acid into ketone bodies about 10 times faster than did perfused livers from fed controls; differences in the rates of ketogenesis from octanoate were, however, less pronounced (<2-fold). The relative rates of ketone body production from (-)-octanoylcarnitine more closely resembled those seen with oleic acid than with octanoate. (+)-Octanoylcarnitine, an inhibitor of carnitine acyltransferase, had no effect on octanoate oxidation but did produce significant and quantitatively similar decreases in the rates of oxidation of both oleic acid and (-)-octanoylcarnitine. The results presented support the postulate that the hepatic carnitine acyltransferase system is under strict dietary and/or hormonal control and that it may constitute a primary site for the regulation of hepatic fatty acid oxidation and ketogenesis.

0184 INVESTIGATION OF THE COMPETITION BETWEEN SOME ORGANIC ACIDS FOR BILIARY EXCRETION. *(E.)* Fischer, E.; Varga, F. (Univ. Med. Sch., Pécs, Hungary). *Arch. Int. Pharmacodyn. Ther.* 209(2):349-357, 1974.

The mutual effect of several organic acids (^{35}S-bromsulphthalein, ^{131}I rose bengal, eosin and amaranth) on their biliary excretion was studied following i.v. injection into male Wistar rats with cannulated bile ducts. Compared to control values, plasma and hepatic bromsulphthalein concentrations were higher when bromsulphthalein excretion was inhibited by rose bengal, eosin or amaranth. Conversely, plasma and hepatic rose bengal, eosin or amaranth concentrations were also higher when their excretion was inhibited by bromsulphthalein. None of the 3 inhibitors altered conjugation of bromsulphthalein and glutathione. These results suggested that competition between the compounds for hepatic excretion should occur at the step of uptake or as a result of the inhibition of the biotransformation of bromsulphthalein. The inhibition of excretion of bromsulphthalein, rose bengal and eosin for one another was competitive whereas that for amaranth was non-competitive. Bromsulphthalein and rose bengal were the most potent inhibitors while amaranth was the weakest. The results support the hypothesis that the biliary organic anion carrier has at least 2 different binding sites or that organic anions may be excreted by more than 1 carrier.

0185 EFFECT OF ECK'FISTULA AND PORTACAVAL TRANSPOSITION ON LIVER REGENERATION. *(E.)* Brolsch, C. E.; Lee, S.; Chandler, J. G.; Flamont, Y. M.; Meyer, W.; Kivelitz, H.; Orloff, M. J. (Univ. Surg. Clin., Dusseldorf, Germany). *Eur. Surg. Res.* 6(8):8, 1974.

0186 INFLUENCE OF X-IRRADIATION ON THE DIFFERENT STAGES OF THE CELL CYCLE IN REGENERATING RAT LIVER. *(E.)* Barbason, H. (Dep. Pathol. Anat., Univ. Liege, Belgium). *Virchows Arch. [Zellpathol.]* 16(4):363-369, 1974.

0187 IN VITRO BINDING OF ^{3}H-ACROLEIN TO REGENERATING RAT LIVER DNA POLYMERASE. *(E.)* Munsch, N.; De Recondo, A. M.; Frayssinet, C. (Inst. Sci. Cancer Res., Villijuif, France). *Experientia* 30(11):1234-1236, 1974.

0188 PYRUVATE KINASE ISOZYME CHANGES IN PARENCHYMAL CELLS OF REGENERATING RAT LIVER. *(E.)* Garnett, M. E.; Dyson, R. D.; Dost, F. N. (Dep. Biochem. Biophys., Oregon State Univ., Corvallis). *J. Biol. Chem.* 249(16):5222-5226, 1974.

0189 THE PREDICTION OF IMMEDIATE SURVIVAL AFTER PORCINE HEPATIC TRANSPLANTATION. *(E.)* Battersby, C.; Egerton, W.; Balderson, G.; Kerr, J. F.; Burnett, W. (Dep. Surg. Pathol., Univ. Queensland, Brisbane, Australia). *Aust. NZ J. Surg.* 44(3):299-307, 1974.

0190 DOSE AND ROUTE OF ALG ADMINISTRATION DEPENDENT EFFECT ON RENAL AND LIVER ALLOGRAFT SURVIVAL IN DOGS. *(E.)* Rowinski, W.; Szmidt, J.; Olszewski, W.; Lukasiewicz, H.; Polanski, P.; Jakubowski, M.; Bruhl, A.; Tupalska, B.; Nielubowicz, J. (Exp. Clin. Med. Cent., Polish Acad. Sci., Warsaw). *Eur. Surg. Res.* 6(1):34, 1974.

0191 ACTIVE ENHANCEMENT OF CANINE LIVER ALLOGRAFTS. *(E.)* Lie, T. S.; Nakano, H.; Bertram, K.; Moritz, B.; Ebata, H. (Med. Fac., Univ. Bonn, Germany). *Eur. Surg. Res.* 6(1):43, 1974.

0192 AUTOTRANSPLANTATION OF HEPATIC SEGMENTS ARTERIALIZED BY DIRECT VASCULAR IMPLANTATION. *(E.)* Goldsmith, H. S.; Chen, W. F.; Alday, E. S.; Kuroda, K. (Jefferson Med. Coll., Philadelphia, Pa.). *J. Surg. Res.* 17(6):411-416, 1974.

0193 IMPEDANCE MEASUREMENT: COMPARATIVE INVESTIGATIONS DURING ORTHOTOPIC LIVER TRANSPLANTATION. *(E.)* Rigler, B.; Lanner, G.; Herrmann, W.; Stenzl, W. (Clin. Surg. Neurosurg., Univ. Graz, Austria). *Eur. Surg. Res.* 6(1):76, 1974.

0194 THE ROLE OF LIVER TRANSPLANTATION IN ACUTE HEPATIC NECROSIS. A COMPARATIVE STUDY OF ORTHOTOPIC AND HETEROTOPIC GRAFTS. *(E.)* Huguet, C.; Bloch, P.; Opolon, P.; Conard, J.; Lavallard, M. C.; Morisot, P. (INSERM, St. Antoine Hosp., Paris, France). *Eur. Surg. Res.* 6(1):32-33, 1974.

0195 HETEROTOPIC AUXILIARY LIVER TRANSPLANTATION IN CIRRHOTIC RATS. *(E.)* Zelder, O.; Paidlick, A.; Jerusalem, C. R. (Dep. Surg., Univ. Marburg/Lahn, Germany). *Eur. Surg. Res.* 6(Suppl. 1):9, 1974.

0196 CIRCULATING NATRIURETIC GLUCAGON (pG1) — EVIDENCE FOR A ROLE IN DEVELOPMENT OF ASCITES. *(E.)* Schwille, P. O.; Bornhof, C.; Scholz, D.; Samberger, N. M. (Dep. Surg., Univ. Erlangen-Nuremberg, Erlangen, Germany). *Eur. Surg. Res.* 6(Suppl. 1): 4, 1974.

0197 THE INFLUENCE OF DIET ON UNCONJUGATED HYPERBILIRUBINAEMIA. *(E.)* Gollan, J. L.; Hatt, K. J.; Billing, B. H. (R. Free Hosp., London, England). *Digestion* 10(4/5):350-351, 1974.

0198 ON THE MODIFICATION OF THE DIVALENT CATION-BINDING BY PHOSPHOLIPIDS IN TUMOURS. *(E.)* Anghileri, L. J. (GHS Clin., Essen, Germany). *Experientia* 30(10):1200-1201, 1974.

0199 IN VITRO ENHANCEMENT OF HEPATIC MICROSOMAL BIPHENYL 2-HYDROXYLATION BY CARCINOGENS. *(E.)* McPherson, F.; Bridges, J. W.; Parke, D. V. (Dep. Biochem., Univ. Surrey, Guildford, England). *Nature* 252(5483):488-489, 1974.

0200 DIFFERENTIAL LECTIN AGGLUTINATION OF FETAL, DIVIDING-POSTNATAL, AND MALIGNANT HEPATOCYTES. *(E.)* Becker, F. F. (New York Univ. Sch. Med., N. Y.). *Proc. Natl. Acad. Sci. USA* 71(11):4307-4311, 1974.

0201 PORTACAVAL SHUNT AND METABOLISM IN THE NORMAL PIG. *(E.)* Hickman, R.; Meyers, O. L.; Engelbrecht, G. H. C.; Keraan, M.; Saunders, S. J.; Terblanche, J. (Dep. Surg., Univ. Cape Town, South Africa). *S. Afr. J. Sci.* 70(9):284, 1974.

0202 PORTACAVAL SHUNT IN THE PIG 1. EFFECT ON SURVIVAL, BEHAVIOR, NUTRITION, AND LIVER FUNCTION. *(E.)* Cuschieri, A.; Baker, P. R.; Holley, M. P.; Hanson, C. (Dep. Surg., Univ. Liverpool, England). *J. Surg. Res.* 17(6):387-396, 1974.

0203 HEPATIC ENCEPHALOPATHY FOLLOWING PORTACAVAL SHUNT IN DOGS: EFFECTS OF AMINO ACID INFUSIONS OF KNOWN COMPOSITION. *(E.)* Fischer, J. E.; Aguirre, A.; Funovics, J.; Yoshimura, N.; Westman, T.; Wesdorp, R. I. C.; Keane, J.; James, J. H. (Massachusetts Gen. Hosp., Boston). *Gastroenterology* 67(4):791, 1974.

0204 CHANGES IN THE CONCENTRATIONS OF ELEMENTS IN LIVER AFTER PARTIAL HEPATECTOMY. *(E.)* Volm, M.; Schumacher, J.; Wayss, K.; Wesch, H. (Inst. Exp. Pathol., Heidelberg, Germany). *Experientia* 30(11):1255-1257, 1974.

0205 RAT LIVER DNA-DEPENDENT RNA NUCLEOTIDYLTRANSFERASES AFTER PARTIAL HEPATECTOMY. *(E.)* Benecke, B. J.; Kruger, C.; Seifart, K. H. (Inst. Physiol. Chem., Univ. Marburg/Lahn, Germany). *Hoppe Seylers Z. Physiol. Chem.* 355(10):1174, 1974.

0206 THE DEPENDENCE OF THE THERAPEUTIC EFFECT ON THE DOSAGE AND THE ANIMALS' FEEDING STATE WHEN APPLYING NUCLEOTIDES AND NUCLEOSIDES IN CASES OF EXPERIMENTAL LIVER DAMAGE. *(E.)* Rau, R. M. (Dep. Surg., Univ. Munster, Germany). *Digestion* 10(4/5):376-377, 1974.

0207 ENZYME ACTIVITY AFTER ACUTE ISCHEMIC LIVER DAMAGE. EXPERIMENTAL STUDIES IN THE PIG. *(E.)* Erhardt, W.; Fischer, M.; Gartmaier, P.; Maier, G.; Pfeiffer, U.; Pichotka, B.; Stoetter, L. (Dep. Surg., Tech. Univ. Munich, Germany). *Digestion* 10(4/5):346-347, 1974.

0208 EVALUATION OF MITOCHONDRIAL FUNCTION IN THE ISCHEMIC RAT. *(E.)* Daniel, A. M.; Beaudoin, J. G. (R. Victoria Hosp., Montreal, Canada). *J. Surg. Res.* 17(1):19-25, 1974.

0209 INFLUENCE OF HYPERVENTILATION ON SERUM POTASSIUM DURING AND AFTER LIVER ISCHEMIA. *(E.)* Bengmark, S.; Hafstrom, L. O. (Goteborg Hosp., Sweden). *Eur. Surg. Res.* 6(1):26-31, 1974.

0210 THE INFLUENCE OF LIVER INJURY ON THE DISTRIBUTION OF DIGITOXIN IN THE RAT. *(E.)* Rosenthal, W. S.; Cagin, N.; Somberg, J. C.; Pitchumoni, C. S.; Levitt, B. (New York Med. Coll., N. Y.). *Arch. Int. Pharmacodyn. Ther.* 209(2):369-373, 1974.

0211 THE EFFECT OF HEPATIC INJURY UPON THE DEVELOPMENT OF AMOEBIC LIVER ABSCESS IN HAMSTERS. *(E.)* Gogler, H.; Knight, R. (London Sch. Hyg. Trop. Med., England). *Ann. Trop. Med. Parasitol.* 68(2):177-185, 1974.

0212 FIBRE-ENTRAPPED ENZYMES: A NEW TOOL FOR THE TREATMENT OF ACUTE LIVER FAILURE? *(E.)* Dioguardi, N.; de Franchis, R.; Mari, D.; Morisi, F.; Marconi, W. (A. Migliavacca Cent. Liver Dis., Univ. Milan, Italy). *Digestion* 10(4/5):344-345, 1974.

0213 AMMONIA AND AMINO ACIDS IN THE BRAIN OF THE RAT WITH CHRONIC HEPATIC FAILURE. *(E.)* Van Leuven, F.; Weyne, J. (Dep. Norm. Pathol. Physiol., Univ. Ghent, Belgium). *Arch. Int. Physiol. Biochem.* 82(4):739-743, 1974.

0214 CHANGES IN THE BLOOD-BRAIN BARRIER IN ACUTE HEPATIC COMA. *(E.)* Livingstone, A. S.; Hinchey, E. J.; Goresky, C. A. (Montreal Gen. Hosp., Canada). *Gastroenterology* 67(4):812, 1974.

0215 HEMODYNAMIC STUDIES OF THE NORMAL DOG LIVER. *(E.)* Kostic, K.; Veljkovic, V.; Susic, D.; Kentera, D. (Fac. Med., Belgrade, Yugoslavia). *Digestion* 10(4/5):361, 1974.

0216 HEMODYNAMIC STUDY OF 3-5 YEARS OLD H-GRAFT PORTACAVAL SHUNTS IN THE DOG. *(E.)* Gillot, C.; Lenriot, J. P.; Baron, M.; Leandri, J.; Peronneau, P.; Monnier, J. P. (Bicetre Hosp., Paris, France). *Eur. Surg. Res.* 6(Suppl. 1):5-6, 1974.

0217 EFFECT OF PORTACAVAL ANASTOMOSIS ON HEPATIC BLOOD FLOW IN THE RAT. *(E.)* Liehr, H.; Grun, M.; Peignoux, M. (Dep. Med., Univ. Wurzburg, Germany). *Digestion* 10(4/5):363, 1974.

0218 THE PORTO-SYSTEMIC COLLATERAL PATTERN IN THE RAT. AN ANGIOGRAPHIC AND ANATOMICAL STUDY AFTER PARTIAL OCCLUSION OF THE PORTAL VEIN. *(E.)* Halvorsen, J. F.; Myking, A. O. (Haukeland Hosp., Univ. Bergen, Sweden). *Eur. Surg. Res.* 6(3):183-195, 1974.

0219 THE USE OF GELATIN TISSUE ADHESIVE IN HEMOSTASIS OF THE RAT LIVER. *(E.)* Weisgerber, G.; Douvin, D.; Huguet, C. (INSERM, St. Antoine Hosp., Paris, France). *Eur. Surg. Res.* 6(1):60-61, 1974.

0220 EFFECT OF HEMODILUTION AND HEMOCONCENTRATION ON HEPATIC BLOOD FLOW AND MEMBRANE POTENTIAL. *(E.)* Lambotte, L. (Lab. Exp. Surg., Louvain, Belgium). *Eur. Surg. Res.* 6(1):25-26, 1974.

0221 THE REAGGREGATION OF ADULT RAT LIVER CELLS MAINTAINED IN VITRO. *(E.)* Alwen, J.; Lawn, A. M. (Lister Inst. Prev. Med., London, England). *Exp. Cell Res.* 89(1):197-205, 1974.

0222 EFFECT OF DIABETES ON THE ULTRASTRUCTURE OF THE HEPATOCYTE AND ON THE DISTRIBUTION AND ACTIVITY OF RIBOSOMES IN THE FREE AND MEMBRANE-BOUND POPULATIONS. *(E.)* Pain, V. M.; Lanoix, J.; Bergeron, J. J. M.; Clemens, M. J. (London Sch. Hyg. Trop. Med., England). *Biochim. Biophys. Acta* 24(4):487-498, 1974.

0223 ORIGIN AND DIVISION OF LIVER MACROPHAGES DURING STIMULATION OF THE MONONUCLEAR PHAGOCYTE SYSTEM. *(E.)* Warr, G. W.; Sljivic, V. S. (St. Mary's Hosp. Med. Sch., London, England). *Cell Tissue Kinet.* 7(6):559-565, 1974.

0224 HISTOCHEMICAL DEMONSTRATION OF CHANGES IN LIVER CELL ENZYMES IN PREGNANT MICE INFECTED WITH COXSACKIE B_3 VIRUS. *(E.)* Lansdown, A. B. G.; Ellaby, S. J. (Clin. Res. Cent., Harrow, England). *Histochemistry* 40(2):175-180, 1974.

0225 FORMATION OF ACTIVE 40 S RIBOSOMAL SUBUNITS IN LIVER IN THE PRESENCE OF AFLATOXIN B_1. *(E.)* Moule, Y.; Sarasin, A. (Inst. Sci. Cancer Res., Villejuif, France). *Chem. Biol. Interact.* 9(1):1-6, 1974.

0226 FETAL HEPATIC DRUG METABOLISM IN THE NON-HUMAN PRIMATE, Macaca arctoides. *(E.)* Dvorchik, B. H.; Stenger, V. G.; Quattropani, S. L. (Pennsylvania State Univ. Coll. Med., University Park). *Drug Metab. Dispos.* 2(6):539-544, 1974.

0227 RELATIONSHIP BETWEEN PLASMA ANTIPYRINE HALF-LIVES AND HEPATIC MICROSOMAL DRUG METABOLISM IN DOGS. *(E.)* Vessell, E. S.; Lee, C. J.; Passananti, G. T.; Shively, C. A. (Milton S. Hershey Med. Cent., Pennsylvania State Coll. Med., Hershey). *Pharmacology* 10(6):317-328, 1973.

0228 METABOLIC N- AND α-C-OXIDATION OF NOREPHEDRINE BY RABBIT LIVER MICROSOMAL FRACTIONS AND SYNTHESIS OF THE METABOLIC PRODUCTS. *(E.)* Beckett, A. H.; Jones, G. R.; Al-Sarraj, S. (Chelsea Coll., Univ. London, England). *J. Pharm. Pharmacol.* 26(12):945-951, 1974.

0229 SELENIUM SUPPLEMENTATION, MATURITY AND SEX DEPENDENCE IN DIETARY NECROTIC LIVER DEGENERATION. *(E.)* Southwell-Keely, P. T.; Johnstone, I. L.; Cole, E. R. (Sch. Wool Pastoral Sci., Univ. New South Wales, Kensington, Australia). *Aust. J. Exp. Biol. Med. Sci.* 52(2):405-408, 1974.

0230 INTRAHEPATIC CHOLESTASIS INDUCED BY 1,3-BIS(2-CHLOROETHYL)-1-NITROSOUREA (BCNU). *(E.)* Thompson, G. R.; Larson, R. E. (Sch. Pharm., Oregon State Univ., Corvallis). *Gastroenterology* 67(4):831, 1974.

0231 EFFECT OF ANTITHYMOCYTE AND ANTILYMPHOCYTE GLOBULIN ON THE PATHOGENESIS OF MURINE HEPATITIS. *(E.)* Perrotto, J. L.; Wands, J. R.; Galdibini, J.; Isselbacher, K. J. (Massachusetts Gen. Hosp., Boston). *Gastroenterology* 67(4):820, 1974.

0232 PREVENTIVE EFFECT OF RIBOFLAVIN 2',3',4', 5'-TETRABUTYRATE ON INACTIVATION OF RAT LIVER MICROSOMAL ENZYMES INDUCED BY CARBON TETRACHLORIDE. *(E.)* Matsuoka, S.; Tahara, K.; Ohama, H. (Tokyo Tanabe Res. Lab., Japan). *J. Nutr. Sci. Vitaminol.* 20(4):333-336, 1974.

0233 EFFECT OF CHRONIC LOW-LEVEL EXPOSURE TO HALOTHANE ON HEPATIC MICROSOMAL ENZYME ACTIVITY. *(E.)* Dudley, A. W.; Allen, J. R.; Chang, L. W.; Dudley, M. A. (Univ. Wisconsin Med. Sch., Madison). *Am. J. Pathol.* 78(1):14a, 1974.

0234 EFFECT OF ACTINOMYCIN D AND CHLORAMPHENICOL ON COPPER RETENTION IN SUBCELLULAR LIVER FRACTIONS OF COPPER INJECTED RATS. *(E.)* Balevska, P. (Inst. Physiol., Acad. Sci., Sofia, Bulgaria). *Dokl. Acad. Nauk (Bulg.)* 27(8):1157-1160, 1974.

0235 METABOLISM OF THIAMPHENICOL AND COMPARATIVE STUDIES OF ITS URINARY AND BILIARY EXCRETION WITH CHLORAMPHENICOL IN VARIOUS SPECIES. *(E.)* Uesugi, T.; Ikeda, M.; Hori, R.; Katayama, K.; Arita, T. (Meiji Coll. Pharm., Tokyo, Japan). *Chem. Pharm. Bull. (Tokyo)* 22(11):2714-2722, 1974.

0236 EFFECT OF PHENOBARBITAL ON THE LEVEL OF GOSSYPOL IN THE LIVER AND THE EFFECT OF GOSSYPOL AND PHENOBARBITAL ON LIVER MICROSOMAL O-DEMETHYLATION AND LIPID PEROXIDATION ACTIVITIES IN THE RAT. *(E.)* Skutches, C. L.; Smith, F. H. (Dep. Anim. Sci., North Carolina State Univ., Raleigh). *J. Nutr.* 104(12):1567-1575, 1974.

0237 EFFECTS OF METHOTREXATE AND PHENOBARBITAL ON THE HEPATIC MICROSOMAL DRUG-METABOLIZING ENZYMES IN NORMAL RAT. *(E.)* Basu, T. K.; Williams, D. C. (Marie Curie Mem. Found., Oxted, England). *Chemotherapy* 21(1):33-40, 1975.

0238 EFFECTS OF PHENOBARBITAL (PB), ETHANOL (E) AND DIETARY FAT ON IN VITRO HEPATIC DRUG METABOLIZING ENZYMES AND THE MICROSOMAL ETHANOL OXIDIZING SYSTEM (MEOS). *(E.)* Joly, J. G.; Hetu, C. (St. Luke Hosp., Montreal, Canada). *Gastroenterology* 67(4):800, 1974.

0239 CHANGES IN HEPATIC RNA SYNTHESIS IN THE STARVE-REFEED RESPONSE OF THE RAT. *(E.)* Pfeifer, G. D.; Szepesi, B. (Agric. Res. Serv., U.S. Dep. Agric., Beltsville, Md.). *J. Nutr.* 104(9): 1178-1184, 1974.

0240 CONCENTRATIONS IN VITRO OF VARIOUS ENZYMES IN LIVER HOMOGENATES DURING THE FIRST FEW DAYS OF A PERIOD OF STARVATION. *(E.)* den Otter, W. (Dep. Pathol., State Univ., Utrecht, Netherlands). *Arch. Int. Physiol. Biochim.* 82(4):611-624, 1974.

0241 EFFECTS OF SUCKLING AND FOOD DEPRIVATION ON THE ACTIVITY OF CITRATE ENZYMES IN THE LIVER OF THE RAT. *(E.)* Alemany, M.; Herrera, E. (Dep. Gen. Physiol., Fac. Sci., Univ. Barcelona, Spain). *Horm. Metab. Res.* 6(4):264-267, 1974.

0242 EFFECT OF DIETARY PROTEIN AND AMINO ACID MIXTURE ON PROTEIN SYNTHESIS IN VITRO IN RAT LIVER. *(E.)* von der Decken, A.; Omstedt, P. T. (Wenner-Gren Inst. Exp. Biol., Stockholm, Sweden). *Nutr. Metab.* 16:325-336, 1974.

0243 EXPERIMENTAL DEFICIENCY OF PLASMA LECITHIN: CHOLESTEROL ACYLTRANSFERASE (LCAT) ACTIVITY BY GALACTOSAMINE. *(E.)* Ragland, J. B.; Kuiken, L.; Sabesin, S. M. (Univ. Tennessee Sch. Med., Memphis). *Gastroenterology* 67(4):821, 1974.

0244 EFFECT OF CORTISOL ON PHOSPHORYLATION OF ACIDIC PROTEINS IN LIVER NUCLEI FROM ADRENALECTOMIZED RATS. *(E.)* Schauder, P.; Starman, B.; Williams, R. H. (Dep. Med., Univ. Washington, Seattle). *Experientia* 30(11):1277-1279, 1974.

0245 STUDIES ON THE METABOLIC INTERRELATIONSHIP BETWEEN BIOTIN AND FOLIC ACID IN RATS. *(E.)* Nair, C. P. P.; Noronhia, J. M. (Bhabha At. Res. Cent., Bombay, India). *J. Nutr. Sci. Vitaminol.* 20(4):243-247, 1974.

0246 ACTIVITY OF MICROSOMAL ENZYMES IN THE LIVERS OF RATS FED RAPESEED OIL. *(E.)* Collomb, M. H.; Albrecht, R.; Griffaton, G.; Manchon, P.; Lowy, R. (Bichat Hosp., Paris, France). *Enzyme* 18(5):300-309, 1974.

0247 DEGRADATION OF HEPATIC TRYPTOPHAN PYRROLASE IN NON-PREGNANT AND PREGNANT RATS. *(E.)* Clark, C. M.; Goodlad, G. A. J. (Dep. Biochem., Univ. St. Andrews, Fife, Scotland). *Enzyme* 18(5): 317-320, 1974.

0248 REGULATION OF SUBSTRATE SPECIFIC FORMS OF UDP-GLUCURONYLTRANSFERASE IN RAT LIVER. *(E.)* Bock, K. W.; Frohling, W.; White, I. N. H. (Inst. Toxicol., Tubingen, Germany). *Digestion* 10(4/5):337, 1974.

0249 REGULATION OF PYRIDOXAL 5'-PHOSPHATE METABOLISM IN LIVER. *(E.)* Li, T. K.; Lumeng, L.; Veitch, R. L. (Indiana Univ. Sch. Med., Indianapolis). *Biochem. Biophys. Res. Commun.* 61(2):677-685, 1974.

0250 HUMAN LIVER AMINOPEPTIDASE. ROLE OF METAL IONS IN MECHANISM OF ACTION. *(E.)* Garner, Jr., C. W.; Behal, F. J. (Texas Tech. Univ. Sch. Med., Lubbock). *Biochemistry* 13(16):3227-3233, 1974.

0251 COMPOSITION OF LIVER HISTONES IN AGING RAT AND MOUSE. *(E.)* Carter, D. B.; Chae, C. B. (Dep. Biochem., Univ. North Carolina, Chapel Hill). *J. Gerontol.* 30(1):28-32, 1974.

0252 INDUCTION OF TYROSINE AMINOTRANSFERASE AND DEPRESSION OF PROTEIN SYNTHESIS IN RAT LIVER BY A TRYPTOPHAN-FREE MIXTURE OF AMINO ACIDS. *(E.)* Pamart, B.; Girard-Globa, A.; Bourdel, G. (Cent. Nutr. Res., Meudon-Bellevue, France). *J. Nutr.* 104(9):1149-1156, 1974.

0253 THE UPTAKE OF LABELLED SULPHUR BY THE PROTEINS OF HUMAN LIVER SLICES INCUBATED WITH L-(^{35}S)-METHIONINE OR (^{35}S)-SULPHATE. *(E.)* Heinonen, K. (Child. Hosp., Univ. Helsinki, Finland). *Life Sci.* 15(3):463-470, 1974.

0254 ADAPTIVE SYNTHESIS OF RAT LIVER FATTY ACID SYNTHETASE: EVIDENCE FOR IN VITRO FORMATION OF ACTIVE ENZYME FROM INACTIVE PROTEIN PRECURSORS AND 4'-PHOSPHOPANTETHEINE. *(E.)* Yu, H. L.; Burton, D. N. (Dep. Microbiol., Univ. Manitoba, Winnipeg, Canada). *Biochem. Biophys. Res. Commun.* 61(2):483-489, 1974.

0255 EFFECT OF DIFFERENT FATTY ACIDS ON GLYCEROLIPID SYNTHESIS IN ISOLATED RAT HEPATOCYTES. *(E.)* Sundler, R.; Akesson, B.; Nilsson, A. (Dep. Physiol. Chem., Univ. Lund, Sweden). *J. Biol. Chem.* 249(16):5102-5107, 1974.

0256 STUDIES ON THE COMBUSTION OF ETHANOL AND ITS INFLUENCE ON LIVER LIPID METABOLISM. *(E.)* Bolmstrand, R.; Kager, L.; Eklof, R.; Lantto, O. (Huddinge Univ. Hosp., Sweden). *Acta Chir. Scand. [Suppl.]* 446:1-22, 1974.

0257 EFFECT OF ACUTE AND PROLONGED ETHANOL ADMINISTRATION ON SEROTONIN METABOLISM AND EXCRETION IN URINE AND BILE OF RATS. *(E.)* Kekki, M.; Pentikainen, P.; Mustala, O. (Dep. Pharmacol., Univ. Helsinki, Finland). *Q. J. Stud. Alcohol* 35(4):1195-1204, 1974.

0258 THE POSSIBLE INVOLVEMENT OF CYTOCHROME b_5 IN THE OXIDATION OF LAURIC ACID BY MICROSOMES FROM KIDNEY CORTEX AND LIVER OF RATS. *(E.)* Sasame, H. A.; Thorgeirsson, S. S.; Mitchell, J. R.; Gillette, J. R. (Natl. Heart Lung Inst., Bethesda, Md.). *Life Sci.* 14(1):35-46, 1974.

0259 EFFECTS OF FASTING ON INTERMEDIATE HEPATIC METABOLISM OF THE RAT. *(E.)* Phan, T.; Bach, A.; Metais, P. (Civil Hosp., Strasbourg, France). *Arch. Int. Physiol. Biochim.* 82(4):82, 603-609, 1974.

0260 INTERACTION OF METHOTREXATE WITH LIPOTROPIC FACTORS IN THE LIVER. *(E.)* Tuma, D. J.; Barak, A. J.; Sorrell, M. F. (Omaha VA Hosp., Nebr.). *Gastroenterology* 67(4):831, 1974.

0261 7α-HYDROXYLATION OF CHOLESTEROL BY RECONSTITUTED SYSTEMS FROM RAT LIVER MICROSOMES. *(E.)* Bjorkhen, I.; Danielsson, H.; Wikvall, K. (Dep. Chem., Karolinska Inst., Stockholm, Sweden). *Biochem. Biophys. Res. Commun.* 61(3):934-941, 1974.

0262 CHOLESTEROL BIOSYNTHESIS IN RAT FOETUS. EFFECT OF DIETARY CHOLESTEROL. *(E.)* Calandra, S.; Montaguti, M.; Quartaroli, G. S. (Inst. Gen. Pathol., Univ. Modena, Italy). *Digestion* 10 (4/5):339-340, 1974.

0263 SYNTHESIS OF SERUM ALBUMIN IN ISOLATED RAT LIVER PERFUSED BY A SYNTHETIC MEDIUM WITH FLUOROCARBON FC-80 EMULSION AS OXYGEN CARRIER. *(E.)* Novakova, V.; Birke, G.; Plantin, L. O. (King Gustaf V Res. Inst., Huddinge, Sweden). *Acta Physiol. Scand.* 92(3):289-302, 1974.

0264 LIVER GLUTATHIONE AND GLUTATHIONE REDUCTASE RESPONSE OF ENDOTOXIN-TREATED MICE. *(E.)* Brown, P. L.; Jeffries, C. D. (Sch. Med., Wayne State Univ., Detroit, Mich.). *Infect. Immun.* 11(1):8-13, 1975.

0265 EFFECT OF HYPOGLYCEMIC SULFONYLUREAS ON HEPATIC FRUCTOSE METABOLISM IN THE RAT. *(E.)* Schonborn, J.; Poser, W.; Panten, U.; Hasselblatt, A. (Inst. Pharmacol. Toxicol., Univ. Gottingen, Germany). *Horm. Metab. Res.* 6(4):284-289, 1974.

0266 THE MECHANISM OF THE NON-OXIDATIVE SEGMENT OF THE PENTOSE CYCLE IN THE LIVER. *(E.)* Rognstad, R.; Katz, J. (Cedars-Sinai Med. Res. Inst., Los Angeles, Calif.). *Biochem. Biophys. Res. Commun.* 61(2):774-781, 1974.

0267 LIVER STORAGE OF VITAMIN A BY RATS FED CARROTS IN VARIOUS FORMS. *(E.)* Sweeney, J. P.; Marsh, A. C. (Agric. Res. Serv., U.S. Dep. Agric., Beltsville, Md.). *J. Nutr.* 104(9):1115-1120, 1974.

0268 REGULATION OF THE MITOCHONDRIAL OXIDATION-REDUCTION STATE IN INTACT HEPATOCYTES BY CALCIUM IONS. *(E.)* Otto, D. A.; Ontko, J. A. (Oklahoma Med. Res. Found., Oklahoma City). *Biochem. Biophys. Res. Commun.* 61(2):743-749, 1974.

0269 THE RELATION BETWEEN URINARY EXCRETION OF TRYPTOPHAN AND NIACIN METABOLITES AND PYRIDINE NUCLEOTIDES IN THE LIVER IN RATS FED A TRYPTOPHAN-DEFICIENT DIET. *(E.)* Ohguri, S. (Inst. Public Health, Tokyo, Japan). *J. Nutr. Sci. Vitaminol.* 20(4):257-261, 1974.

0270 LIVER MICROSOMAL ELECTRON TRANSPORT SYSTEMS. III. THE INVOLVEMENT OF CYTOCHROME b_5 IN THE NADPH-SUPPORTED CYTOCHROME P-450-DEPENDENT HYDROXYLATION OF CHLOROBENZENE. *(E.)* Lu, A. Y. H.; Levin, W.; Selander, H.; Jerina, D. M. (Dep. Biochem. Drug Metab., Hoffman-La Roche Inc., Nutley, N. J.). *Biochem. Biophys. Res. Commun.* 61(4):1348-1355, 1974.

0271 HEPATOCYTE SUSPENSIONS AS A MODEL FOR DEMONSTRATION OF DRUG HEPATOTOXICITY. *(E.)* Zimmerman, H. J.; Kendler, J.; Libber, S.; Lukacs, L. (VA Hosp., Washington, D. C.). *Biochem. Pharmacol.* 23(15):2187-2189, 1974.

0272 A COMPARISON OF THREE PHENOL METHODS FOR EXTRACTION OF RAT LIVER RNA. *(E.)* Paus, P. N. (Dep. Microbiol., Univ. Oslo, Norway). *Acta Chem. Scand.* 28(7):797-801, 1974.

0273 PITFALLS IN THE CORRECTION OF MICROSOMAL LOSSES BY CYTOCHROME P-450. *(E.)* Joly, J. G.; Pesant, Y.; Doyon, C. (St. Luke Hosp., Montreal, Canada). *Gastroenterology* 67(4):801, 1974.

0274 METABOLISM OF LITHOCHOLIC ACID IN MAN. *(E.)* Cowen, A. E.; Korman, M. G.; Hofmann, F. (Mayo Clin., Rochester, Minn.). *Gastroenterology* 67(4):785, 1974.

0275 HYDROXYLATIONS OF BILE ACIDS BY RECONSTITUTED SYSTEMS FROM RAT LIVER MICROSOMES. *(E.)* Bjorkhem, I.; Danielsson, H.; Wikvall, K. (Dep. Chem., Karolinska Inst., Stockholm, Sweden). *J. Biol. Chem.* 249(20):6439-6445, 1974.

0276 IMPAIRED BILIARY LECITHIN-CHOLESTEROL SECRETION: A CONSEQUENCE OF DEFICIENT DIETARY CHOLINE. *(E.)* Robins, S. J. (VA Hosp., Boston, Mass.). *Gastroenterology* 67(4):823, 1974.

0277 COMPARED ACTIONS OF SECRETIN (S), CHOLECYSTOKININ (CK) AND CAERULEIN (C) ON DOG CHOLERESIS. *(E.)* Angelucci, L.; Catalani, A.; Tavoloni, N. (Inst. Pharmacol. Pharmacognosy, Univ. Rome, Italy). *Br. J. Pharmacol.* 52(3):461, 1974.

0278 BILE SALT INDEPENDENT BILE FLOW DURING RELEASE AND DRAINAGE OF COMMON BILE DUCT IN CHOLECYSTECTOMIZED DOGS. *(E.)* Morris, T. Q.; Baker, K. J.; Bradley, S. E. (Coll. Physicians Surg., Columbia Univ., New York, N. Y.). *Gastroenterology* 67(4):817, 1974.

0279 EFFECT OF CHOLECYSTECTOMY AND SPHINCTEROTOMY ON BILE ACID WASHOUT IN THE DOG. *(E.)* von Bergmann, K.; Schultheiss, H. R.; Paumgartner, G.; Preisig, R. (Dep. Clin. Pharmacol., Univ. Bern, Switzerland). *Gastroenterology* 67(4):833, 1974.

0280 ACTION OF THE CHRONIC IRRITATION OF THE SECTION OF THE VAGO-SYMPATHETIC NERVES UPON THE CHOLEDOCHAL SPHINCTER IN THE DOGS. *(E.)* Sarles, J. C.; Devaux, M. A.; N'Guyen, R.; Michel, G. (Res. Unit Dig. Pathol., INSERM, Marseille, France). *Eur. Surg. Res.* 6(Suppl. 1):55-56, 1974.

0281 ACRYLAMIDE GEL ELECTROPHORESIS OF LIVER PROTEIN DURING EXTRAHEPATIC CHOLESTASIS IN THE RAT. *(E.)* Lundborg, H.; Blomstrand, C.; Hamberger, A. (Sahlgrenska Hosp., Univ. Goteborg, Sweden). *Eur. Surg. Res.* 6(3):168-175, 1974.

0282 THE EFFECT OF PHENOBARBITAL ON BILE ACID KINETICS IN MAN. *(E.)* Schiff, E. R.; Parker, I. T.; Dick, R. D. (VA Hosp., Miami, Fla.). *Gastroenterology* 67(4):828, 1974.

0283 MICROBIAL BILE ACID TRANSFORMATION. *(E.)* Midtvedt, T. (Rikshosp., Univ. Oslo, Norway). *Am. J. Clin. Nutr.* 27(11):1341-1347, 1974.

0284 TRANSIENT BILIARY LIQUID CRYSTALS AND METASTABLE SUPERSATURATION IN PRAIRIE DOGS DURING RAPID DIETARY INDUCTION OF CHOLESTEROL GALLSTONES. *(E.)* Holzbach, R. T.; Corbusier, C.; Marsh, M. (Cleveland Clin. Found., Ohio). *Gastroenterology* 67(4):799, 1974.

0285 CHANGES IN HEPATOBILIARY PERMEABILITY DURING SODIUM TAUROCHOLATE (Na-TC) LOADING IN NORMAL RATS (N) AND IN RATS TREATED WITH ETHINYL ESTRADIOL (EE). *(E.)* Herz, R. G.; Bradley, S. E. (Coll. Physicians Surg., Columbia Univ., New York, N.Y.). *Gastroenterology* 67(4):797, 1974.

See also, 0023, 0026, 0300, 0302, 0307, 0590, 0591.

0286 THE PERICRYPTAL FIBROBLAST SHEATH. IV. REPLICATION, MIGRATION, AND DIFFERENTIATION OF THE SUBEPITHELIAL FIBROBLASTS OF THE CRYPT AND VILLUS OF THE RABBIT JEJUNUM. *(E.)* Parker, F. G.; Barnes, E. N.; Kaye, G. I. (Coll. Physicians Surg., Columbia Univ., New York, N.Y.). *Gastroenterology* 67(4):607-621, 1974.

Epithelial-mesenchymal relations were examined in light microscopic studies of human and rabbit small intestine and in electron microscopic and autoradiographic studies of the pericryptal (subepithelial) sheath in the rabbit jejunum. Under the light microscope human and rabbit jejunum displayed the same arrangement of subepithelial fibroblasts with small collagen fibers encircling the crypts and villi. For autoradiographic studies, each of 22 adult American Dutch rabbits received a total of 5 mCi ^{3}H-thymidine as 5 i.v. injections spaced 30 min apart; a pair of rabbits were sacrificed at selected intervals over 21 days. Initial uptake of ^{3}H-thymidine by epithelial cells and fibroblasts was confined to the crypt, and both jejunal populations replicated and migrated through the crypt-villus in approximate synchrony. The initial labeling ratio of epithelial cells to subepithelial fibroblasts was 5 times as large as that reported for the rabbit colon. Electron microscopy showed that fibroblasts of the jejunal sheath were fewer, larger, and more widely spaced than those in the colon. Moreover, the well-developed collagen table seen in the colon was not present in the jejunum. In this respect, the relations among epithelium, fibroblast, and venous capillaries resembles more closely those in the gallbladder than in the colon. Differences in the colonic pericryptal fibroblast sheath and the jejunal subepithelial fibroblast sheath may be related to differences in their absorptive functions. This study demonstrates the importance of interacting epithelial-mesenchymal systems in tissues of adult animals. Additional investigations are necessary to understand the basis and regulation of these interactions in normal and neoplastic tissue.

0287 THE GUT-ASSOCIATED LYMPHOID SYSTEM: NATURE AND PROPERTIES OF THE LARGE DIVIDING CELLS. *(E.)* Guy-Grand, D.; Griscelli, C.; Vassalli, P. (Necker Child. Hosp., Paris, France). *Eur. J. Immunol.* 4(6):435-443, 1974.

The lymphoid cells and ^{3}H-thymidine labeled cells of the Peyer's patches, mesenteric lymph nodes and thoracic duct lymph of Balb/c mice and rats were studied by combined immunofluorescence and autoradiography, using antisera directed against T (mouse-specific lymphocyte antigen) or B (mouse-specific bone marrow-derived lymphocyte antigen) cell antigenic determinants, or against various immunoglobulin (Ig) chains. In some experiments, mice were immunized by 3 weekly injections in the footpads with ferritin (0.5 mg); the mice were killed 4 days after the last injection. Other mice were immunized by injection of 15 x 10^{6} sheep RBC in each footpad on the 5th, 4th, and 3rd day before sacrifice. Rats were immunized with killed *Bacillus pertussis*. The migration pattern of these various cell populations after transfer to syngeneic mice or rats was determined by radioactivity counting and autoradiography. Labeled cells from mesenteric lymph nodes and thoracic duct lymph had, in contrast to labeled cells from the peripheral lymph nodes, a marked tendency to localize in the gut, and they migrated in a graft of fetal intestine as well as in the recipient's own gut. This indicates that this selective accumulation is not the result of a mechanism recognizing antigen absorbed from the gut lumen. B-specific labeled cells localizing in the gut contained surface IgA and intracellular IgA, which transform into IgA plasma cells in the lamina propria. It is suggested that the progenitors of the intestinal IgA plasma cells are proliferating surface IgA cells from the germinal centers of labeled Peyer's patches cells. These IgA cells must migrate *via* lymphatics to mesenteric lymph nodes and thoracic duct lymph and mature during this migration, acquiring intracellular IgA chains as well as a gut-localizing mechanism which might be related to this increased IgA synthesis. T-specific labeled cells localizing in the gut migrated both in the lamina propria and in the intestinal epithelium. Most, if not all, of the intraepithelial lymphocytes of the gut mucosa appear to be T lymphocytes derived from rapidly dividing cells. Two types of T-specific labeled cells may exist in the lymphoid system: one that is circulating and localizes in the gut, and a second that does not localize in the gut and may be sessile.

0288 BEHAVIOR OF BILIARY PHOSPHOLIPIDS IN INTESTINAL LUMEN DURING FAT DIGESTION IN RAT. *(E.)* Nalbone, G.; Lairon, D.; LaFont, H.; Domingo, N.; Hauton, J.; Sarda, L. (Res. Group Lipid Transp. U 130, INSERM, Marseille, France). *Lipids* 9(10): 765-770, 1974.

The behavior of biliary phospholipids in the small intestinal lumen of male Wistar rats was studied at different stages of lipolysis to investigate their possible role in phenomena connected with intestinal absorption of triglycerides. Separation of the lipid constituents from total intraluminal contents of 4 rats receiving 800 mg triolein (stomach intubation) showed that at the beginning of lipolysis (1 hr after ingestion) all lecithins, cholesterol, and bile salts were eluted within the 1st column volume in highly opalescent fractions containing triglycerides, diglycerides, and small amounts of free fatty acids and monoglycerides. Intraluminal content analysis (4 rats) at an intermediate lipolysis stage (75 min postingestion) showed 75% of the biliary phospholipids and cholesterol being eluted with the 1st column volume along with triglycerides, diglycerides, lipase, and colipase. These fractions contained 33% of the free fatty acids, 14% of the monoglycerides, and 26% of the bile salts eluted from the column. Intraluminal analysis (4 rats) at the end of lipolysis (90 min after ingestion of 300 mg triolein) resulted in 10% lecithin, 2% monoglyceride, 14% fatty acid, and 6% bile salt elutions along with small amounts of triglycerides and diglycerides in the 1st column volume. The major part of the bile lecithins, cholesterol, bile salts, and lipolysis products was

eluted in the 2nd volume (clear fractions). *In vitro* chromatographic studies of reconstituted intraluminal contents confirmed these separation patterns. The experiments demonstrate that biliary lecithins are associated to fatty acids, monoglycerides, and bile salts in the aqueous phase.

0289 INFLUENCE OF PERIODICITY OF EATING ON INTESTINAL GLYCOLYTIC ENZYMES IN THE RAT. *(E.)* Romsos, D. R.; Leveille, G. A. (Dep. Food Sci. Human Nutr., Michigan State Univ., East Lansing). *Comp. Biochem. Physiol.* [A] 49(3):533-540, 1974.

The effects of meal-eating patterns of intestinal hexokinase and pyruvate kinase activities, intestinal mucosal weight, and on diurnal fluctuations of plasma free fatty acids and insulin levels were studied in 60 male Sprague-Dawley rats. Half the animals were fed *ad lib* ("nibbler"), a diet consisting of 58, 30 and 12% carbohydrate, protein and fat, resp., while the other 30 rats were fed for 2 hr in the morning only ("meal eaters") for 5 weeks. Nibblers weighed about 25-40% more ($P<0.05$) and ate significantly more (158 gm/week *vs* 143 gm/week) than the meal eaters, indicating that meal eaters are more efficient than nibblers in converting food to body weight gain. Mucosal weight was greater in the upper small intestine than in the lower segment in both nibblers and meal eaters ($P<0.05$). Hexokinase activity was higher in meal eaters than in nibblers whereas pyruvate kinase activity was about the same. Within intestinal segments, however, pyruvate kinase activity was higher in the upper segment of both groups; hexokinase activity was higher in the lower segment. Total upper intestinal hexokinase activity was significantly greater in meal-fed rats at 4 pm and 9 pm while lower intestinal hexokinase activity was greater in meal eaters only at 4 pm. Hexokinase specific activities, however, were the same at all times. Pyruvate kinase activity was significantly greater in meal eaters only in the upper segment at 4 pm. Plasma free fatty acid and insulin levels showed variations related to meal intake patterns in both nibblers and meal eaters. The results do not support the existence of a time-conditioned response for hexokinase in rats.

0290 DIPEPTIDASE ACTIVITY IN THE SMALL INTESTINAL MUCOSA DURING PREGNANCY AND LACTATION IN THE RAT. *(E.)* Rolls, B. A. (Natl. Inst. Res. Dairy., Shinfield, England). *Br. J. Nutr.* 33(1):1-9, 1975.

Hooded Norwegian rats were mated and at weekly intervals during pregnancy and lactation and after weaning, the dipeptidase activity was determined in supernatant fractions of small intestinal mucosal homogenates for 2 dipeptides: glycyl-L-leucine dipeptidase and L-alanyl-L-glutamic acid dipeptidase. The mucosal supernatant contained 87.1% and 96.1% of the total mucosal glycyl-L-leucine and L-alanyl-L-glutamic acid dipeptidase activities, resp. Slight to moderate increases in total supernatant dipeptidase activities occurred during pregnancy with increases to over 200% of baseline values during the 2nd week of lactation. A similar pattern was observed for dipeptidase specific activity suggesting that the increases in enzyme activity were real and not merely a result of mucosal hyperplasia. Six females which failed to feed their offspring did not show changes in dipeptidase activities. The increases in dipeptidase activities were correlated more closely to increases in mucosal nitrogen content than to increased food intake during pregnancy and lactation and could have been due to hormonal stimulation.

0291 THE ROLE OF VASCULAR AND LYMPH CAPILLARIES IN THE CLEARANCE OF INTERSTITIALLY INJECTED ALBUMIN IN ACUTE AND CHRONIC INFLAMMATION OF THE COLON. *(E.)* Sheppard, M. S.; Sterns, E. E. (Dep. Surg., Queen's Univ., Kingston, Canada). *Surg. Gynecol. Obstet.* 139(5):707-711, 1974.

The role of the vascular and lymphatic capillaries in the clearance of interstitially-injected albumin from the colon during the acute stage of sterile inflammation and in 8 weeks of recovery was investigated in 8 normal mongrel dogs. Within the first 5 hr, 20% of radioiodinated albumin injected (1 ml with an activity ranging from 50-500 μCi/ml) into the interstitial space of the colon was returned to the circulation by the thoracic duct. During the same period, 2% of albumin was transported directly by the blood. Acute sterile inflammation was induced by subserosal injection of sterile talc in saline. Dogs were studied at 4, 7, 21, and 56 days after talc injection. Both lymph flow and the amount of interstitially injected albumin increased by approximately 50% during 5 hr. There was direct access to the blood by slightly over 4% of the interstitially injected dosage, and most of this occurred immediately after injection. Further vascular accumulation was slight and in the same order as in the control group. The changes with respect to albumin transport were most obvious 7 days after the onset of inflammation and subsided rapidly, so that function returned to near normal levels within 3 weeks.

0292 INTESTINAL METABOLISM OF LIPIDS AND LIPOPROTEINS. *(E.)* Gangl, A.; Ockner, R. K. (Univ. California Sch. Med., San Francisco). *Gastroenterology* 68(1):167-186, 1975.

0293 THE MECHANISM OF THE HUMAN INTESTINAL SUCRASE ACTION. *(E.)* Zagalak, B.; Curtius, H. C. (Inst. Pediatr., Univ. Zurich, Switzerland). *Biochem. Biophys. Res. Commun.* 62(3):503-509, 1975.

0294 THE ORIGIN OF ANTIBODY IN INTESTINAL SECRETION OF SHEEP. *(E.)* Husband, A. J.; Lascelles, A. K. (Dairy Res. Unit, Univ. Farms, Camden, Australia). *Aust. J. Exp. Biol. Med. Sci.* 52(5): 791-799, 1974.

0295 POSSIBLE ANTIMICROBIAL FUNCTION OF THE LARGE MOLECULAR SIZE VITAMIN B_{12}-BINDING PROTEIN. *(E.)* Gullberg, R. (Karolinska Hosp., Stockholm, Sweden). *Scand. J. Gastroenterol.* [*Suppl.*] 9(29):19-21, 1974.

0296 ARE ENTERIC ENDOTOXINS ABLE TO ESCAPE FROM THE INTESTINE? *(E.)* Gans, H.; Matsumoto, K. (New York Hosp.-Cornell Med. Coll., N.Y.). *Proc. Soc. Exp. Biol. Med.* 147(3):736-739, 1974.

0297 INTRACELLULAR CHLORIDE ACTIVITIES IN THE MUCOSAL EPITHELIUM OF RABBIT TERMINAL ILEUM. *(E.)* Henriques de Jesus, C.; Ellory, J. C.; Smith, M. W. (Inst. Anim. Physiol., Cambridge, England). *J. Physiol. (Lond.)* 244(1):31P-32P, 1975.

0298 RESISTANCE OF ENDOGENOUS INTRALUMINAL PHOSPHOLIPIDS TO PANCREATIC HYDROLYSIS IN THE INTACT RAT. *(Fr.)* Olsson, O.; Boucrot, P. (U.E.R. Nutr., Dijon, France). *Arch. Int. Physiol. Biochim.* 82(4):679-699, 1974.

0299 THE SITES OF INHIBITION OF α-METHYLGLUCOSIDE TRANSFER IN HAMSTER INTESTINE BY L-PROLINE AND GALACTOSE. *(E.)* Browne, J. L.; Smyth, D. H. (Middlewood Hosp., Sheffield, England). *J. Physiol. (Lond.)* 244(1):29P-30P, 1975.

0300 PROPANOLOL INHIBITS BILE ACID STIMULATION OF ADENYLATE CYCLASE IN COLONIC MUCOSA. *(E.)* Coyne, M. J.; Bonorris, G. G.; Chung, A.; Goldstein, L. I.; Schoenfield, L. J. (Cedars-Sinai Med. Cent., Los Angeles, Calif.). *Gastroenterology* 67(4):786, 1974.

0301 KINETIC STUDY OF ELECTROSTATIC INTERACTIONS BETWEEN INTESTINAL ALKALINE PHOSPHATASE AND ITS SUBSTRATE IN CALVES. *(Fr.)* Julien, M.; Ouellet, L. (Dep. Chem., Laval Univ., Quebec, Canada). *Can. J. Chem.* 52(17):3087-3097, 1974.

0302 SOURCES OF CHOLESTEROL IN THE INTESTINAL LYMPH IN RATS FED A CHOLESTEROL-FREE DIET. *(E.)* Ho, K. J.; Taylor, C. B. (Univ. Birmingham, Med. Cent., Alabama). *Proc. Soc. Exp. Biol. Med.* 147(3):826-829, 1974.

0303 TOPOLOGICAL STUDIES ON THE HYDROLASES BOUND TO THE INTESTINAL BRUSH BORDER MEMBRANE. 1. SOLUBILIZATION BY PAPAIN AND TRITON X-100. *(E.)* Louvard, D.; Maroux, S.; Vannier, C.; Desnuelle, P. (Cent. Biochem. Mol. Biol., Marseilles, France). *Biochim. Biophys. Acta* 375(2):236-248, 1975.

0304 ILEAL EPITHELIAL CELL MIGRATION IN ACUTE RENAL FAILURE. AUTORADIOGRAPHIC STUDIES IN THE MOUSE. *(E.)* McDermott, F. T.; Dalton, M. K.; Galbraith, A. J. (Alfred Hosp., Monash Univ., Melbourne, Australia). *Am. J. Dig. Dis.* 19(12):1116-1119, 1974.

0305 CELL RENEWAL IN EXPERIMENTALLY OBSTRUCTED SMALL INTESTINE IN RAT. *(E.)* Kamei, H.; Watanabe, T.; Takemitsu, T.; Momoi, T.; Sugimoto, K.; Kondo, T. (Nagoya Univ. Sch. Med., Japan). *Jpn. J. Surg.* 3(4):237-247, 1973.

0306 CECAL FISTULA FORMATION AND CECECTOMY IN THE RABBIT. *(It.)* Angelini, A.; Cioffi, L. A.; Proto, V. (Inst. Hum. Physiol. I., Univ. Naples, Italy). *Boll. Soc. Ital. Biol. Sper.* 49(23):1390-1396, 1973.

See also, 0012, 0027, 0032, 0507.

GENERAL

0307 CHYLOUS ASCITES CAUSED BY LYMPHATIC AND PORTAL STENOSIS: EXPERIMENTAL STUDY IN THE RAT. *(Fr.)* Leymarios, J.; Roze, C.; Souchard, M.; Debray, C. (Bichat Hosp., Paris, France). *Biol. Gastroenterol. (Paris)* 7(1):53-59, 1974.

Chylous ascites was produced in nearly 100% of 80 male Wistar rats by ligation of 2 intestinal lymph trunks and subtotal stenosis of the portal vein produced by ligation with a metal rod 7 mm in diameter followed by removal of the rod. All rats were fed regular diets until sacrificed in groups of ten at intervals up to 10 days after operation. From day 2 to 7, 1-20 ml of light red chylous ascitic fluid was obtained from nearly every rat; the fluid contained lipid and triglyceride levels of the same order of magnitude or greater than serum levels (measured from aortic blood in 10 control rats) but significantly less than intestinal lymph levels (measured in a 24-hr collection of intestinal lymph from 7 control rats). On day 10 less than 3 ml/animal was obtained from 5 of the 10 rats and none from the others. Two other operations, isolated ligation of 2 lymph trunks (50 rats) and isolated partial stenosis of the portal vein (50 rats), were ineffective in producing chylous ascites. In the former group, there was no peritoneal fluid at all except in 2 rats with 2 ml of fluid of very high lipid and triglyceride content, apparently leaked from intestinal lymph. In the latter group, only about 20% sacrificed on days 1, 5, and 7 had any ascites; 80% killed on day 3 had clear ascites with very low lipid and triglyceride content and less than 4 ml in volume. The success of the combined

operation is attributed at least in part to the effect of portal hypertension in preventing the escape of lymph through lympho-venous connections following ligation of lymph trunks.

0308 EFFECTS OF A HIGH-BEEF DIET ON BOWEL FLORA: A PRELIMINARY REPORT. *(E.)* Maier, B. R.; Flynn, M. A.; Burton, G. C.; Tsutakawa, R. K.; Hentges, D. J. (Univ. Missouri Sch. Med., Columbia). *Am. J. Clin. Nutr.* 27(12):1470-1474, 1974.

A study was made to determine whether the quantitative and/or qualitative bowel flora of man could be perceived as changing as a function of a controlled, systematic dietary sequence which included a high-beef regimen. Ten healthy male volunteers (aged 21-30 yr) were used. Four diets were compared, each administered for 4 weeks. The first, an initial normal diet, consisted in g/day of: protein, 77.075; fat, 56.78; CHO, 443.86; fiber, 5.78; and ash, 20.02. A meatless diet contained, in g/day: protein, 85.35; fat, 77.36; CHO, 430.71; fiber, 4.86; and ash, 19.71. A high-meat diet was comprised of, in g/day: protein, 178.73; fat, 74.94; CHO, 436.04; fiber, 6.50; and ash, 20.07. The closing normal diet contained, in g/day: protein, 83.32; fat, 64.24; CHO, 446.68; fiber, 5.71; and ash, 20.07. Total anaerobic counts were elevated during the high-meat and final normal diets, but the differences were not statistically significant. *Bacteroides* species decreased from a value of 2×10^{10} to 7×10^{9} organisms/g dry weight during the meatless diet regimen. Both the high-meat and the final normal diets caused *Bacteroides* to be elevated to almost 10^{11} organisms/g. *Fusobacteria* increased during meatless and high-meat diets. *Propionibacteria*, *Eubacteria*, and *Bifidobacteria* were stable throughout. *Clostridia* was depressed by the high-meat diet. Data indicate that *Bacteroides* and *Bifidobacteria* have an inverse population relation. These analyses indicated that the genus *Bacteroides* may be statistically elevated under high-meat diets, while the average coliform population was elevated during the meatless diet and yet did not drop under the effect of a high-meat regimen.

0309 ADRENERGIC MECHANISMS IN THE CANINE GASTRIC CIRCULATION. *(E.)* Zinner, M. J.; Kerr, J. C.; Reynolds, D. G. (Walter Reed Army Inst. Res., Washington, D. C.). *Physiologist* 17(3):366, 1974.

0310 DRUG-INDUCED CHANGES IN CAPILLARY FILTRATION COEFFICIENT AND BLOOD FLOW IN THE INNERVATED SMALL INTESTINE OF THE ANAESTHETIZED CAT. *(E.)* Richardson, P. D. I. (Med. Coll. St. Bartholomew's Hosp., London, England). *Br. J. Pharmacol.* 52(4):481-498, 1974.

0311 AUGMENTATION OF ARTERIAL HEPATIC AND RENAL FLOW BY EXTRACTED AND SYNTHETIC PARATHYROID HORMONE. *(E.)* Charbon, G. A.; Hulstaert, P. F. (Dep. Cardiovasc. Surg., Univ. Utrecht, Netherlands). *Endocrinology* 95(2):621-626, 1974.

0312 SPECIAL PROBLEMS ASSOCIATED WITH THE ISOLATION AND IDENTIFICATION OF INTESTINAL BACTERIA IN FECAL FLORA STUDIES. *(E.)* Moore, W. E. C.; Holdeman, L. V. (Coll. Agric. Life Sci., Virginia Polytech. Inst., Blacksburg). *Am. J. Clin. Nutr.* 27(12):1450-1465), 1974.

0313 SPECIFIC IMMUNE ANTIBACTERIAL MECHANISMS IN THE INTESTINES OF MICE. *(E.)* Rowley, D. (Dep. Microbiol., Univ. Adelaide, Australia). *Am. J. Clin. Nutr.* 27(12):1417-1423, 1974.

0314 METABOLIC INTERACTIONS AMONG INTESTINAL MICROORGANISMS. *(E.)* Wolin, M. J. (Dep. Dairy Sci. Microbiol., Univ. Illinois, Urbana). *Am. J. Clin. Nutr.* 27(11):1320-1328, 1974.

0315 INTERACTIONS BETWEEN MECHANISMS CONTROLLING THE INTESTINAL MICROFLORA. *(E.)* Freter, R. (Dep. Microbiol., Univ. Michigan, Ann Arbor). *Am. J. Clin. Nutr.* 27(12):1409-1416, 1974.

0316 INTESTINAL ANTIBODY SECRETION IN THE YOUNG PIG IN RESPONSE TO ORAL IMMUNIZATION WITH Escherichia coli. *(E.)* Porter, P.; Kenworthy, R.; Noakes, D. E.; Allen, W. D. (Unilever Res., Sharnbrook, England). *Immunology* 27(5):841-854, 1974.

0317 METABOLISM OF DRUGS BY MICROORGANISMS IN THE INTESTINE. *(E.)* Goldman, P.; Peppercorn, M. A.; Goldin, B. R. (Harvard Med. Sch., Boston, Mass.). *Am. J. Clin. Nutr.* 27(11):1348-1355, 1974.

0318 BIOHYDROGENATION OF STEROLS AND FATTY ACIDS BY THE INTESTINAL MICROFLORA. *(E.)* Eyssen, H.; Parmentier, G. (Rega Inst., Univ. Louvain, Belgium). *Am. J. Clin. Nutr.* 27(11):1329-1340, 1974.

0319 SOME ASPECTS OF THE METABOLISM OF CECAL MICROFLORA IN PROTEIN NUTRITION IN THE RABBIT. *(It.)* Coppola, S.; Percuoco, G.; Proto, V. (Inst. Agric. Microbiol., Univ. Naples, Italy). *Boll. Soc. Ital. Biol. Sper.* 49(21):1253-1259, 1973.

0320 QUANTITATIVE VARIATIONS IN THE INTESTINAL MICROFLORA OF RABBITS FOLLOWING LIGATION OF THE PANCREATIC DUCT. *(Fr.)* Catala, J.; Bonnafous, R. (Lab. Physiol. Dig. Nutr., Paul Sabatier Univ., Toulouse, France). *C. R. Acad. Sci. [D] (Paris)* 278(26):3351-3353, 1974.

0321 STUDY OF A NEW CLASS OF Escherichia coli MUTANTS WHICH ARE RESISTANT TO COLICINS D AND E_3. *(Fr.)* Hamon, Y.; Peron, Y. (Pasteur Inst., Paris, France). *C. R. Acad. Sci. [D] (Paris)* 279(10):867-870, 1974.

0322 THE EFFECTS OF ENDOTOXEMIA AND BACTEREMIA ON GASTROINTESTINAL DRUG ABSORPTION IN MICE AND RATS. *(E.)* Hurwitz, A.; Furtado, D.; Low, R. R. (Univ. Kansas Med. Cent., Kansas City). *J. Pharmacol. Exp. Ther.* 192(1):236-241, 1975.

0323 DIFFERENCES IN RENAL EXCRETION OF SUB-GROUPS OF BILE ACIDS DEPENDING ON THE INTRODUCTION OF POLAR GROUPS. *(E.)* Back, P. (Med. Clin., Univ. Freiburg, Germany). *Digestion* 10(4/5): 322-323, 1974.

0324 DYNAMICS OF THE AMINO ACID METABOLISM IN ANIMALS WITH ONE STOMACH UNDER VARIOUS PERFORMANCE CONDITIONS. *(Ger.)* von Wiesemueller, W. (Dep. Zootech., Univ. Rostock, Germany). *Wissenschaftl. Z. Univ. Rostock, Math. Naturwissenchaftl. Reihe* 22(8):899-929, 1973.

0325 IMMUNOLOGICAL POTENCY AND STABILITY OF NATIVE AND SYNTHETIC SECRETINS. *(E.)* Boden, G.; Dinoso, V. P.; Owen, O. E. (Temple Univ. Health Sci. Cent., Philadelphia, Pa.). *Gastroenterology* 67(6):1119-1125, 1974.

0326 CONTROL OF FOOD INGESTION IN RUMINANTS BY DUODENAL CHEMORECEPTORS, *(Fr.)* Bueno, L.; Ruckebusch, Y. (Natl. Vet. Sch., Toulouse, France). *C. R. Acad. Sci. [D] (Paris)* 279(5):409-412, 1974.

0327 STUDIES OF ANTIINFLAMMATORY AGENTS. XXV. EFFECTS OF ANTI-INFLAMMATORY AGENTS ON LEUKOCYTE MIGRATION IN CARRAGEENIN-PERITONITIS OF RATS. *(Jpn.)* Nakanishi, M.; Goto, K. (Res. Lab., Yoshitomi Pharm. Ind., Ltd., Fukuoka, Japan). *J. Pharm. Soc. Jpn.* 94(10):1218-1223, 1974.

0328 CELL MEMBRANE PERMEABILITY CHANGE: AN IMPORTANT STEP IN HORMONE ACTION. *(E.)* Petersen, O. H. (Inst. Med. Physiol. C, Univ. Copenhagen, Denmark). *Experientia* 30(10):1105-1107, 1974.

DIAGNOSTIC PROCEDURES

0329 SERUM ALPHA-FETOPROTEIN (AFP) IN BENIGN AND MALIGNANT GASTROINTESTINAL DISEASES: EVALUATION OF AN IMMUNOENZYMATIC ASSAY. *(E.)* Alpert, E. (Harvard Med. Sch., Boston, Mass.). *Clin. Chim. Acta* 58(1):77-83, 1974.

A sensitive and specific quantitative immunoenzymatic assay was developed for α-fetoprotein. α-Fetoprotein levels were over 50 ng/ml in 78% of 79 patients with primary liver carcinoma. Serum concentrations ranged from 50 to 7,000,000 ng/ml with no correlation to any clinical or pathological features. Of 71 who were tested 14 (20%) were HB Ag-positive by hemagglutination inhibition and an additional 20 of 71 (28%) were HB Ab-positive by hemagglutination. Five children (aged 3 months to 4 yr) with hepatoblastoma had serum α-fetoprotein concentrations between 250 ng/ml and 1.3 mg/ml. In 19 patients with metastatic carcinoma of the liver only 2 were positive. Of 54 patients with acute viral hepatitis at the peak of disease, 7 (13%) showed α-fetoprotein levels between 50 and 1000 ng/ml. Of 9 patients with massive hepatic necrosis probably due to viral hepatitis, 4 were positive. In 86 patients with various forms of chronic liver disease, α-fetoprotein was detected in levels between 50 and 1000 ng/ml in 4 of 17 with both HB Ag-positive and HB Ag-negative chronic hepatitis (23.5%). Of 69 patients with chronic non-viral disease including active post-necrotic and Laennec's cirrhosis, and primary biliary cirrhosis, only 1 patient was positive. No α-fetoprotein was detected in patients with hepatic hemangioendothelium, lymphoma limited to the liver and spleen, hepatic hemangioma, extra-hepatic biliary tract malignancies (5 cases), primary malignancies outside the gastrointestinal tract (17), active inflammatory bowel disease (17), non-viral acute hepatitis (24, including patients with alcoholic hepatitis, halothane hepatitis, and thorazine induced hepatitis), and patients in the stage of active liver regeneration following partial hepatectomy (6). It is concluded that α-fetoprotein elevations in nonmalignant liver disease are not due solely to hepatic regeneration but appear related to viral injury. The immunoenzymatic assay used does not require purified antigen or radioisotope equipment and can detect and quantitate clinically significant α-fetoprotein levels greater than 50 ng/ml.

0330 ALIMENTARY TRACT METASTASES FROM MALIGNANT MELANOMA. *(E.)* Beckly, D. E. (United Bristol Hosp., England). *Clin. Radiol.* 25(3):385-389, 1974.

During the period of 1960 to 1972, 3 men (aged 31-88 yr) presented with combinations of malaise, anorexia, weight loss, epigastric discomfort, shortness of breath, vomiting and lethargy. In each case the only obviously diseased site was the alimentary tract, and there was no clinical evidence of more widespread lesions. Chest radiograms were normal. Each patient had a history of malignant melanoma. The small bowel showed intraluminal filling defects in 2 cases with central collections of barium in 1. The other patient showed a large filling defect in the stomach, with a barium collection in a central depression. Diagnosis of metastatic melanoma of the bowel was obtained at autopsy in 2 of these cases, and by biopsy of the lesions in the 3rd. In view of the relatively large differential diagnosis of the single lesion, definite diagnosis is not likely to be forthcoming from barium studies alone. In these cases biopsy will be necessary. However, examination of the small bowel should be performed. If the lesions are multiple and relevant history of a previous malignant melanoma is available, the gastrointestinal lesions may be suspect as metastases. It is then advisable to conduct a carful search for metastases at other sites, including liver and brain scans.

0331 DIAGNOSIS OF BARRET'S ESOPHAGUS WITH RADIOISOTOPES. *(E.)* Gordon, F.; Ramirez-Degollado, J.; Munoz, R.; Cuaren, A.; Landa, L. (Gen. Hosp., Natl. Med. Cent., Mexico City, Mexico). *Am. J. Roentgenol. Radium Ther. Nucl. Med.* 121(4):716-719, 1974.

A diagnostic procedure using radioisotopes, based on selective concentration of Tc^{99m} pertechnetate by the ectopic gastric mucosa found in Barrett's esophagus, was used in a group of 15 patients with no upper gastrointestinal tract disease. A second group of 7 patients suspected of having Barrett's esophagus was studied by the same method. In all cases of suspected Barrett's esophagus, 5 mCi Tc^{99m} pertechnetate was administered endovenously without previous preparation. In all normal cases, the images showed a uniform concentration of the radioisotope by the gastric mucosa, with a clear limitation of the profile of the organ. In all cases of Barrett's esophagus, apart from the normal concentration in the stomach, concentration of the radioisotope was also observed in the distal esophagus, corresponding to the location of the ectopic gastric mucosa. In every case the scintigraphic findings had good correlation with previous roentgenologic and endoscopic findings and the findings noted at surgery. Pertechnetate ion replaces the anions of elements of Group VII, and when introduced into the body, is accumulated by the thyroid, the salivary glands and the gastric mucosa as well as by other organs, but not by the esophagus. The radiation dose absorbed by the patient is minimal with this technique, and permits the use of Tc^{99m} in amounts ample enough to produce a large number of counts of radioactivity, which generally improves the diagnostic efficiency of the method. It was concluded that diagnosis of Barrett's esophagus with Tc^{99m} pertechnetate is a fast, easy, reliable and innocuous method that may be used as a complementary procedure in the study of patients suspected of having this disease.

0332 THE ENDOSCOPIC DIAGNOSIS OF GASTRIC LYMPHOMA. GROSS CHARACTERISTICS AND HISTOLOGY. *(E.)* Nelson, R. S.; Lanza, F. L. (M.D. Anderson Hosp., Houston, Tex.). *Gastrointest. Endosc.* 21(2):66-68, 1974.

Endoscopic evaluation was made of 40 patients with gastric lymphoma by both color photography and histological examination, the latter obtained by guided

gastroscopic biopsy and brush cytology. Of the 40 patients, 30 had reticulum cell sarcoma; 7, lymphocytic lymphoma; 1, Hodgkin's disease; and 2 were unclassified. Of the 40, 21 neoplasms were primary in the stomach and 19 had gastric involvement as part of generalized disease. A slight improvement in gross diagnosis was noted over a previous series, but the sole distinctive characteristic was a peculiar "volcano-like" ulcer crater noted in 9 of 30 patients with reticulum cell sarcoma. A correct diagnosis was made by gastroscopy in 13 of 21 in the primary lymphoma group and by radiology in 7 of 21. In generalized lymphoma, gastroscopy was diagnostic in 17 of 19 and radiology in 14 of 19. An incorrect diagnosis of carcinoma in 7 cases each by gastroscopy and radiology was the most conspicuous and consistent error. Benign ulceration was mistaken in 1 case by gastroscopy and 4 by radiology and gastritis was diagnosed in 1 patient each by both methods. Gastroscopic photography was helpful for a retrospective study of specific features of various lesions but did not improve diagnosis in the frequently confusing situation presented by infiltrations which could be diagnosed either as carcinoma or lymphoma. It was a deciding feature in the initial recognition of the volcano-like ulcer noted in reticulum cell sarcoma. Direct biopsy yielded 5 false-negatives, for an accuracy of 87%. Cytology was performed in 20 of the cases in 4 of whom neoplastic cells were obtained on smear (20% accuracy). Cytology should be useful in the occasional case in which biopsy might be negative and cytology positive. The general conclusion is that gastroscopy with vigorous biopsy attempts should be carried out in all cases of suspected gastric lymphoma as a means of early and nonoperative histologic diagnosis.

0333 GASTROGRAFIN IN PERFORATED DUODENAL ULCER AND ACUTE PANCREATITIS. *(E.)* Fraser, G. M.; Fraser, I. D. (Leith Hosp., Edinburgh, Scotland). *Clin. Radiol.* 25(3):397-402, 1974.

Gastrografin (50 ml p.o.) was given to all cases of clinically suspected perforated duodenal ulcer over a 4 yr period 5 min before the chest and abdominal films were taken. Gastrografin was similarly given to any case of suspected acute pancreatitis. A total of 51 patients were investigated. Correct diagnoses were obtained in 24 of the 25 cases of perforated duodenal ulcer and in 9 of 10 cases of acute pancreatitis. Of the 25 perforated ulcer cases (21 male and 4 female aged 18-79 yr), radiological findings included 17 cases (68%) showing free gas and 24 (96%) showing either free gas or a leak of gastrografin or both. The one undiagnosed case showed no free gas nor a leak of gastrografin and was found at operation to have a sealed off perforation. The time interval between perforation and x-ray examination is important in determining whether free intraperitoneal gas is seen. All 7 patients in this series who showed no free gas but who had a leak of gastrografin had a relatively short time interval (mean 2 1/2 hr), whereas the mean for the entire series was 8 hr. The 10 acute pancreatitis patients were all males (aged 31-73 yr). Radiological findings in patients with pancreatitis with gastrografin included stretching of the duodenal loop (8 cases), Frostberg's inverted 3 sign (1), upward displacement of the stomach (8), and localized indentation of the gastric antrum (2). This method is viewed as a simple, safe and quick method of substantially improving diagnostic accuracy in cases of perforated duodenal ulcer and in cases of acute pancreatitis.

0334 DIRECT VISION BRUSHING CYTOLOGY IN THE DIAGNOSIS OF COLO-RECTAL MALIGNANCY. *(E.)* Bemvenuti, G. A.; Prolla, J. C.; Kirsner, J. B.; Reilly, R. W. (Dep. Med., Univ. Chicago, Ill.). *Acta Cytol. (Baltimore)* 18(6):477-481, 1974.

Direct vision brushing exfoliative cytology was performed in 135 patients using conventional proctoscopy and in 67 patients using colonoscopy. Twenty-three patients had proven colo-rectal malignancy; the remaining 179 had non-malignant disease. Cytology alone identified 19 of the malignancies, an accuracy of 82.5%. Biopsy alone identified 13 malignancies, an accuracy of 68.4%. Using a combination of procedures all patients with malignancies had a correct diagnosis established by at least 1 of the 2 procedures. Thus, this combination of methods yielded no false-negatives in this series. The high detection rate and the complete safety of brushing cytology emphasize its clinical utility, especially in chronic ulcerative colitis. Where biopsy is contraindicated, brushing cytology under direct vision may remain the only selective early sampling method for tissue diagnosis.

0335 ENDOSCOPIC RETROGRADE CHOLANGIOPANCREATOGRAPHY IN THE DOG: A MODEL FOR TRAINING AND RESEARCH. *(E.)* Falkenstein, D. B.; Abrams, R. M.; Kessler, R. E.; Jones, B.; Johnson, G.; Zimmon, D. S. (Manhattan VA Hosp., New York, N.Y.). *Gastrointest. Endosc.* 21(1):25-26, 1974.

A canine model for training and research in endoscopic retrograde cholangiopancreatography is described. Mongrel dogs (30-40 pounds), receiving only water for 24-36 hr prior to endoscopy, are given i.v. administrations of atropine (0.4 mg/25 pound) and Surital (sodium thiamylal, 7.5 mg/pound) prior to laryngeal intubation. With the dog in the left lateral recumbent position on a fluoroscopy table, a side-viewing Olympus JF-B duodenoscope is introduced into the esophagus through a mouth gag. The canine gastric configuration is similar to that of man and presents familiar problems in endoscope manipulation. Passage of the endoscopic head through the pylorus requires accurate placement with attention paid to the size of the loop created by the endoscope within the atonic stomach. Positioning the endoscope in the canine duodenum requires 80-100 cm of endoscope length, closely simulating adult human working distances. Within 3-5 cm of the pylorus, the first of 2 papillae is found on the posterio-medial duodenal wall, appearing as a 1.5-2.0 mm pancake-like whitish excrescence pointing distinctly caudad. A second small papilla is found on the medial duodenal wall 1-2 cm caudad. With the viewing port of the endoscope positioned close to the papilla, the orifice is

cannulated with a catheter prefilled with Hypaque 50% (sodium diatrizoate). There is usually a small pancreatic common channel that bifurcates into the limbs of the canine pancreas, and insertion of the catheter past this bifurcation (more than 3-4 mm) may result in selective filling of 1 pancreatic limb. Fluoroscopy and spot filming allow visualization and documentation of filled duct systems. Duodenal papillae were identified in 7 of 10 animals, and retrograde visualization of the pancreatic or biliary ducts was achieved in 4 dogs. Failures resulted from poor animal preparation, excessive animal size (inability to reach the duodenum in a 60 pound dog), and manipulative fumbling.

0336 NEEDLE ASPIRATION BIOPSY. A SAFE DIAGNOSTIC PROCEDURE FOR LESIONS OF THE PANCREAS. *(E.)* Kline, T. S.; Neal, H. S. (Lankenau Hosp., Philadelphia, Pa.). *Am. J. Clin. Pathol.* 63(1):16-19, 1975.

A review of patients who had had a total of 28 thin-needle pancreatic biopsies taken during laparotomy was undertaken. Of the 28 lesions examined, 18 were malignant and 16 of these were diagnosed unequivocally by cytology. The remaining 10 patients whose lesions were diagnosed as benign, are apparently free of pancreatic malignancy, the majority having been followed for as long as 2 yr. In those patients with malignancy, aspiration biopsy commonly revealed easily identifiable tumor cells. These formed small cell clusters composed of 2-5 cells with marked nuclear anisocytosis and eosinophilic macronucleoli. Other abnormalities included rather scant, sometimes vacuolated, cytoplasm, thickened nuclear membranes, and nuclei containing clumps of chromatin. The benign cells usually exfoliated in large sheets, with polarized cells containing uniform, often eccentric, bland nuclei with moderate cytoplasm. Needle aspiration biopsy of the pancreas at laparotomy is a most useful procedure, both because of the difficulty in recognition of malignancy within this organ and because of the complications of biopsies by conventional technics. If necessary, aspirates may be obtained from multiple areas of the pancreas. Diagnostic accuracy appears high. Biopsy through the duodenum is an added safety precaution for, if a fistula should develop, it would drain directly into the duodenum rather than into the peritoneal cavity.

0337 CRITICAL STUDIES OF THE DIAGNOSTIC VALUE OF CHYMOTRYPSIN DETERMINATIONS IN PANCREATIC DISEASE. *(Ger.)* Brugmann, E.; Rohde, L.; Towe, J.; Teichmann, W. (Med. Clin., Univ. Rostock, Germany). *Dtsch. Z. Verdau. Stoffwechselkr.* 33(4/5):223-225, 1973.

The chymotrypsin levels were determined in feces from different types of patients by the method described by Haverback *et al.* to assess the diagnostic value of chymotrypsin determination in pancreatic diseases. The chymotrypsin levels determined in fecal samples from 64 patients with gastrointestinal involvement, Angina pectoris vasomotorica and other extraabdominal conditions but with intact pancreas ranged from 130 to 5,400 µg/g, with a geometric mean of 1,010 µg/liter. The geometric mean value of the chymotrypsin level was 80 µg/g (10-393 µg/g) in 18 patients with chronic pancreatitis. The chymotrypsin levels were between 5 and 12 µg/g in 5 patients with carcinoma of the pancreas, and consistently below 10 µg/g in 2 patients with the pancreas extirpated. The lower limit of the normal range was calculated to be at 388 µg/g. The high probability of correct diagnosis makes the chymotrypsin determination suitable as a screening test for pancreatic insufficiency.

0338 ARTERIOGRAPHY IN CHRONIC PANCREATITIS. *(Fr.)* L'Hermine, C. (Munic. Hosp., Lille, France). *Ann. Chir.* 27(6):651-655, 1973.

In 15 of 183 cases of chronic pancreatitis, the diagnosis was made on the basis of the arteriogram, which revealed a stricture of the splenic vein. This sign seemed to appear earlier, more frequently, and perhaps more consistently than arterial bruit. The arteriogram was extremely useful in assessing lesions and in detecting certain complications. In 89 cases there were venous lesions, most frequently of the splenic vein. Involvement may also be limited to the splenomesenteric venous trunk, in which case it is useful to examine the integrity of the superior mesenteric vein by a study of the mesenteric arteriogram, since it shows venous return. Superior mesenteric arteriography is the only means of seeing if the involvement is chiefly in the superior mesenteric vein. Splenic complications were relatively uncommon (6 cases), but they were clearly shown by arteriography as a segmentary splenic infarct or localized necrosis or, in more severe cases, as intrasplenic hematoma. One intrahepatic pseudocyst caused by a hematoma was also observed, manifested on the arteriogram as a compressed left branch of the hepatic artery with elongated branches. There were 3 cases of complete obstruction of large arterial trunks: 1 of the splenic artery, which was apparently iatrogenic, and 2 of the hepatic artery, which could have been either iatrogenic or caused by the pancreatitis. Stenosis of the mouth of the celiac trunk occurred in 25 cases. Aortography showed them very clearly, but it was not possible to distinguish between those caused by the pancreatitis and those due to other causes.

0339 LIVER SCANNING IN PATIENTS WITH SUSPECTED ABDOMINAL TUMOR. *(E.)* Fee, Jr., H. J.; Prokop, E. K.; Cameron, J. L.; Wagner, Jr., H. N. (Johns Hopkins Hosp., Baltimore, Md.). *JAMA* 230 (12):1675-1677, 1974.

A series of 70 patients who were to undergo laparotomy for probable intra-abdominal tumors was evaluated using recent technical advances to determine the accuracy of liver scanning as the sole preoperative screening test for hepatic metastases. Ten-view liver and spleen scans were performed as part of routine presurgical evaluation 10 min after i.v. administration of 3 to 5 mCi of technetium Tc 99m sulfur colloid. Of the 70 liver scans, 59 (84%) correctly showed either the presence or absence of me-

tastatic diseases as confirmed by surgery. However, of the 48 eventually proved free of hepatic metastases at surgery, 6 had abnormal scans, for a 13% incidence of false-positive results. Among the 22 patients proved at surgery to have hepatic metastases, 5 had normal scans, a 23% incidence of false-negative results. It is noted that in 2 of these 5 cases the lesions were less than 1 cm in size. The accuracy of the serum alkaline phosphatase levels was 63% with a 46% incidence of false positive and an 18% incidence of false-negative results. It is noted that, in spite of its inaccuracies, the liver scan is probably the best of all single diagnostic tools available to the clinician. However, the accuracy of liver scanning is too low to use it as the routine screening method to detect liver metastases. Unless it is combined with other diagnostic tests, the results of liver scan can be misleading and thus of dubious value in preoperative evaluations of the patient with possible hepatic metastases.

0340 HALF-CLEARANCE TIME OF ^{198}AU COLLOID AS A DISCRIMINANT OF LIVER ABSCESS AND MALIGNANCY. *(E.)* Bahk, Y. W. (St. Mary's Hosp., Seoul, Korea). *Br. J. Radiol.* 47(564):857-861, 1974.

The ability of the half clearance time of ^{198}Au colloid to discriminate among abscess, hepatoma, and metastatic tumor of the liver was investigated in a statistical study. The clinical material consisted of 84 cases of amebic abscess, 33 of pyogenic abscess, 81 of hepatoma, and 61 of metastatic tumor. The half-clearance time discriminated correctly 47.9% of abscesses and 64.2% of hepatomas when the 2 diseases were presented together. Discrimination was correct in 55.7% of metastatic tumors and 80.2% of hepatomas when these 2 malignant tumors were presented as a homogeneous sample. Discrimination of abscess (25.6%) and metastatic tumors (1.6%) presented jointly was very poor. The results indicate that measurement of half-life time, which is simple to perform and does not incur any substantial increase in cost and time, can be used as an ancillary test to discriminate abscess and malignant tumors of the liver, particularly metastatic tumors and hepatoma. When used with other liver function tests in the light of pertinent clinical information, this test may prove beneficial.

0341 THE USES AND LIMITATIONS OF RADIOISOTOPES IN THE INVESTIGATION OF GASTROINTESTINAL DISEASES. *(E.)* Braunstein, P.; Song, C. S. (New York Univ. Med. Cent., N. Y.). *Am. J. Dig. Dis.* 20(1):53-90, 1975.

0342 BREATH ANALYSIS: GASTROENTEROLOGICAL APPLICATIONS. *(E.)* Hepner, G. W. (Pennsylvania State Univ. Sch. Med., Hershey). *Gastroenterology* 67(6):1250-1256, 1974.

0343 RECENT DEVELOPMENTS IN GASTROINTESTINAL ENDOSCOPY. *(E.)* Raskin, J. B. (Univ. Miami Sch. Med., Fla.). *Postgrad. Med.* 57(1):85-96, 1975.

0344 FIBERENDOSCOPY OF THE GASTROINTESTINAL TRACT. *(Fr.)* Faye, C. (Paris Hosp., France). *Gaz. Hop.* 145(15):665-674, 1973.

0345 ACUTE GASTROINTESTINAL BLEEDING: A ONE-YEAR PROSPECTIVE STUDY. *(Ger.)* Kapp, F.; Baerlocher, C.; Fahrlander, H. (Med. Dep., Univ. Basel, Switzerland). *Schweiz. Med. Wochenschr.* 104(45):1609-1613, 1974.

0346 ENDOSCOPIC DIAGNOSIS OF BLEEDING IN THE ESOPHAGUS, STOMACH, AND DUODENUM. *(Ger.)* Junghanns, K.; Seufert, R. (Surg. Clin., Univ. Heidelberg, Germany). *Langenbecks Arch. Chir.* 337:505-510, 1974.

0347 INTERPRETATIONS OF SOME TESTS OF UPPER ALIMENTARY FUNCTION. *(E.)* Hunt, J. N. (Guy's Hosp. Med. Sch., London, England). *Rend. Gastroenterol.* 6(2):139-147, 1974.

0348 ENDOSCOPIC DIAGNOSIS OF BLEEDING IN THE SMALL AND LARGE INTESTINE AND RECTUM. *(Ger.)* Fruhmorgen, P.; Zeus, J.; Demling, L. (Med. Clin., Univ. Erlangen, Germany). *Langenbecks Arch. Chir.* 337:557-561, 1974.

0349 EARLY, PRESYMPTOMATIC, PREINVASIVE DIAGNOSIS OF GASTRIC CARCINOMA. *(It.)* Zanelotti, G. (no affil.). *Med. Soc.* 23(5):208-211, 1973.

0350 SIGNIFICANCE OF GASTROFIBROSCOPIC EXAMINATION IN PATIENTS AFTER STOMACH RESECTION. *(Sl.)* Beno, I. (Res. Inst. Nutr., Bratislava, Czechoslovakia). *Lek. Obzor* 22(6):287-290, 1973.

0351 DIFFERENTIAL DIAGNOSIS OF THE ENDOSCOPICALLY DYED LESIONS BY GASTROSCOPIC CLOSE-UP APPEARANCES. *(E.)* Suzuki, S.; Groitl, H.; Suzuki, H.; Endo, M.; Takemoto, T.; Nakayama, K. (Tokyo Women's Med. Coll., Japan). *Endoscopy* 6(2): 99-104, 1974.

0352 THE EFFECT OF METOCLOPRAMIDE (PRIMPERAN) DURING GASTROSCOPIC OBSERVATION OF THE ANTRUM AND THE PYLORUS. *(E.)* Hradsky, M.; Stockbrugger, R. (Falu Hosp., Falun, Sweden). *Gastrointest. Endosc.* 21(2):55-57, 1974.

0353 B-GLUCURONIDASE IN GASTRIC SECRETIONS. *(E.)* Ayulo, J. A.; Forrest, G. L. (Dep. Med., Univ. California, Irvine). *Am. J. Gastroenterol.* 62(4):337-344, 1974.

0354 DIAGNOSIS OF THE SMALL INTESTINE. *(Ger.)* Eggerth, G. (Dep. Gastroenterol., Vienna, Austria). *Wien. Z. Inn. Med.* 54(7):291-294, 1973.

0355 A NEW POSSIBILITY FOR ROUTINE X-RAY EXAMINATION OF THE SMALL INTESTINE. *(Ger.)* Stallamor, K. (Rudolfstiftung Hosp., Vienna, Austria). *Wien. Z. Inn. Med.* 54(7):299-305, 1973.

0356 A "FALSE-POSITIVE" ABDOMINAL SCAN FOR MECKEL'S DIVERTICULUM. *(E.)* Lentle, B. C.; Scott, G. W. (Dr. W. W. Cross Cancer Inst., Edmonton, Canada). *Br. J. Radiol.* 48(565):59-61, 1975.

0357 COMPARISON OF METHODS TO DIAGNOSE DEFICIENCY OR MALABSORPTION OF VITAMIN B_{12}. *(E.)* Carlmark, B.; Reizenstein, P. (Karolinska Hosp., Stockholm, Sweden). *Scand. J. Gastroenterol.* [*Suppl.*] 9(29):39-42, 1974.

0358 COLONOSCOPY: DEVELOPMENT, TECHNIQUE, INDICATIONS. *(Ger.)* Seyfried, H. (Dep. Gastroenterol., Vienna, Austria). *Wien. Z. Inn. Med.* 54(7):295-298, 1973.

0359 DIASTATIC SEROSAL LACERATIONS: AN UNRECOGNIZED COMPLICATION OF COLONOSCOPY. *(E.)* Livstone, E. M; Cohen, G. M.; Troncale, F. J.; Touloukian, R. J. (Yale Univ. Sch. Med., New Haven, Conn.). *Gastroenterology* 67(6):1245-1247, 1974.

0360 MANOMETRIC STUDIES ON ANORECTAL CONTINENCE IN CHILDHOOD. PART I: METHOD, REFLEX MECHANISMS, CONTINENCE FACTORS. *(Ger.)* Holschneider, A. M.; Metzler, E. M. (Pediatr. Clin., Univ. Munich, Germany). *Bruns Beitr. Klin. Chir.* 221(1): 14-24, 1974.

0361 FUNCTIONAL DIAGNOSIS OF CHRONIC PANCREATIC DISEASES. *(Ger.)* Brunner, H. (Dep. Gastroenterol., Vienna, Austria). *Wien. Z. Inn. Med.* 54(7):287-290, 1973.

0362 SUCCESSFUL MODIFICATIONS FOR PANCREATIC IMAGING. *(E.)* Chandra, S.; Prezio, J. A. (Mercy Hosp., Buffalo, N. Y.). *J. Nucl. Med.* 15(11): 935-937, 1974.

0363 DIAGNOSIS OF EXOCRINE INSUFFICIENCY OF THE PANCREAS. *(Ger.)* Reichmann, J.; Drogies, I. (Altenburg Hosp., Germany). *Dtsch. Z. Verdau. Stoffwechselkr.* 33(1):25-30, 1973.

0364 EXPERIENCES WITH A NEW TEST OF EXOCRINE PANCREATIC FUNCTION IN PROTEIN DEFICIENT PATAS MONKEYS. *(E.)* Gyr, K.; Wolf, R. H.; Imondi, A. R.; Felsenfeld, O. (Univ. Med. Clin., Basel, Switzerland). *Schweiz. Med. Wochenschr.* 104(50): 1888-1889, 1974.

0365 THE SELECTIVE ANGIOGRAPHY OF TR. COELIACUS IN DISEASES ORIGINATING FROM THE PANCREAS. *(E.)* Baldzijski, A.; Mincev, M. (I.P. Pavlov Higher Med. Inst., Plovdov, Bulgaria). *Folia Med. (Plovdov)* 15(4):195-201, 1973.

0366 ENDOSCOPIC RETROGRADE CHOLEDOCHOPANCREATOGRAPHY. *(E.)* Leach, R. E.; Riley, J. C.; Davis, Jr., W. D. (Ochsner Clin., New Orleans, La.). *South. Med. J.* 68(1):49-54, 1975.

0367 DIAGNOSTIC VALUE OF ROUTINE LIVER TESTS. *(E.)* Winkel, P.; Ramsoe, K.; Lyngbye, J.; Tygstrup, N. (Rigshosp., Univ. Copenhagen, Denmark). *Clin. Chem.* 21(1):71-75, 1975.

0368 ENZYMATIC EVALUATION OF HEPATOBILIARY DISEASE. *(E.)* Batsakis, J. G.; Sodeman, T. A.; Deegan, M. J. (Univ. Michigan Med. Cent., Ann Arbor). *Lab. Med.* 5(10):33-37, 40-43, 46-49, 1974.

0369 NON-ENZYME HEPATIC FUNCTION TESTS. *(E.)* Hartwell, R. M.; Hew, Jr., A. Y. (Hotel Dieu, New Orleans, La.). *Lab. Med.* 5(10):24-25, 28-30, 32, 1974.

0370 CLINICAL CORRELATION OF HEPATIC FLOW STUDIES. *(E.)* Witek, J. T.; Spencer, R. P. (Yale Univ. Sch. Med., New Haven, Conn.). *J. Nucl. Med.* 16(1):71-72, 1974.

0371 BILE PLEURITIS: ANOTHER COMPLICATION OF PERCUTANEOUS LIVER BIOPSY. *(E.)* Dosik, M. H. (Univ. California Cent. Health Sci., Los Angeles). *Am. J. Dig. Dis.* 20(1):91-93, 1975.

0372 PROSPECTS AND LIMITATION FOR DIAGNOSIS OF WILSON'S DISEASE FROM LIVER BIOPSIES. *(Ger.)* Brandt, G. (Inst. Pathol., Univ. Erlangen, Germany). *Beitr. Pathol.* 152(2):151-162, 1974.

0373 LIVER SCINTIGRAPHY WITH A SCINTILLATION CAMERA. COMPARISON BETWEEN ^{99m}Tc-Sn-COLLOID AND ^{99m}Tc-PHYTATE. *(Jpn.)* Aburano, T.; Ueno, K.; Watanabe, H.; Hisada, K. (Sch. Med., Kanazawa Univ., Japan). *Radioisotopes* 23(7):391-394, 1974.

0374 AN EVALUATION OF ^{99m}Tc-SULFUR COLLOID LIVER SCINTISCANS AND THEIR USEFULNESS IN METASTATIC WORKUP: A REVIEW OF 1,424 STUDIES. *(E.)* Lunia, S.; Parthasarathy, K. L.; Bakshi, S.; Bender, M. A. (Roswell Park Mem. Inst., Buffalo, N. Y.). *J. Nucl. Med.* 16(1):62-65, 1974.

0375 THE VALUE OF COLLOID AND LIVER CELL SCINTIGRAPHY AS WELL AS LAPAROSCOPY AND LIVER SEROLOGY IN THE DIAGNOSIS OF LIVER TUMORS. *(Ger.)* Muller, J. (Inst. Med. Radiol., Univ. Basel, Switzerland). *Schweiz. Med. Wochenschr.* 104(31):1085-1091, 1974.

0376 HEPATOSPLENIC SCINTIGRAPHY FOR LIVER TUMORS IN CHILDREN. *(Fr.)* Piepsz, A.; Struyven, J.; Decostre, P.; Maurus, R. (St. Pierre Hosp., Brussels, Belgium). *Helv. Paediatr. Acta* 29(4):371-382, 1974.

0377 CLINICAL APPROACH TO JAUNDICE. *(E.)* Dusol, Jr., M.; Schiff, E. R. (Univ. Miami Sch. Med., Fla.). *Postgrad. Med.* 57(1):118-128, 1975.

0378 RADIOLOGICAL CHANGES IN LEFT LOBE AMOEBIC LIVER ABSCESSES. *(E.)* Ramachandran, S. (Colombo North Gen. Hosp., Ragama, Sri Lanka). *Postgrad. Med. J.* 50(589):689-693, 1974.

0379 PSEUDOTUMORS IN ACUTE HEPATITIS. *(E.)* Winston, M. A.; Shapiro, M. (VA Wadsworth Hosp. Cent., Los Angeles, Calif.). *J. Nucl. Med.* 15(11):1039-1040, 1974.

0380 TECHNETIUM 99m SULFUR COLLOID SPLEEN/LIVER RATIO AND OTHER LIVER FUNCTION TESTS IN THE DIAGNOSIS OF CIRRHOSIS. *(E.)* Habibian, M. R.; Kutka, N.; Wilkinson, Jr., R. H.; Goodrich, J. K.; Harris, C. C. (Duke Univ. Med. Cent., Durham, N. C.). *South. Med. J.* 68(1):5-12, 1975.

0381 COMBINED COLLOID AND ROSE BENGAL LIVER SCANNING IN A PATIENT WITH CIRRHOSIS AND A FUNCTIONAL HEPATOMA. *(E.)* Gamlen, T. R.; Ackery, D. M.; Grant, R. W.; Jacoby, R. J.; Kenny, R. W.; MacIver, A. G.; Triger, D. R.; (Southampton Univ. Hosp., England). *Br. J. Radiol.* 48(565):61-62, 1975.

0382 COMPARISON BETWEEN THE ^{131}I-ROSE BENGAL RADIOISOTOPIC HEPATOGRAM INDICES AND THOSE OF OTHER LABORATORY EXAMINATIONS IN PATIENTS WITH CIRRHOSIS HEPATIS. *(E.)* Mincev, M.; Zaharieva, Z. (I. P. Pavlov Med. Inst., Plovdiv, Bulgaria). *Folia Med. (Plovdiv)* 16(1):35-41, 1974.

0383 SCINTIGRAPHIC STUDIES OF THE HEPATIC CIRRHOSES. *(E.)* Bozduganov, A.; Maistorski, A.; Janev, P. (I. P. Pavlov Med. Inst., Plovdiv, Bulgaria). *Folia Med. (Plovdiv)* 15(4):187-194, 1973.

0384 THE SCINTIGRAPHIC DEMONSTRATION OF BILE LEAKAGE UTILIZING ^{131}I-ROSE BENGAL. *(E.)* Wiener, S. N.; Vyas, M. (Mt. Sinai Hosp. Cleveland, Ohio). *J. Nucl. Med.* 15(11):1044-1046, 1974.

0385 GALLIUM GALLBLADDER SCANNING IN CHOLECYSTITIS. *(E.)* Waxman, A. D.; Siemsen, J. K. (Univ. South. California Med. Cent., Los Angeles). *J. Nucl. Med.* 16(2):148-150, 1975.

See also, 0177, 0397, 0427, 0502, 0506, 0521, 0527, 0552, 0561, 0562, 0565, 0580, 0595, 0642, 0671, 0683, 0685, 0696, 0697, 0788.

GASTROINTESTINAL DISEASES

ESOPHAGUS

0386 GASTROINTESTINAL MANIFESTATIONS OF STEINERT'S DISEASE. *(Fr.)* Cappoen, J. P.; Lhermine, C.; Proye, C.; Dumont, J. C.; Voiment, Y. M.; Linquette, M. (Munic. Hosp., Lille, France). *Lille Med.* 18(10):1215-1222, 1973.

Observations of gastrointestinal transit are reviewed in 9 patients with myotonia atrophica. Three patients had high dysphagia associated with pharyngeal (1 case) and/or retrosternal (3 cases) blocks. In 2 cases there were more severe problems in swallowing with attacks of coughing during eating; in one case, these problems were the immediate cause of death. Manometric and cineradiographic studies confirmed that problems in swallowing resulted from a reduction of cricopharyngeal sphincter tonus. In all 9 cases there was esophageal paralysis with few or no peristaltic contractions: this was associated with esophageal dilatation. There was no correlation between the extent of esophageal motor disorders and signs of gastrointestinal dysfunction. The involvement of the esophagus is proof of the involvement of smooth muscle in the disease, since the lower 2/3 of the esophagus is essentially made up of smooth muscle fibers. The stomach, gall bladder, small intestine, and colon may also be involved, but such involvement has not often been demonstrated functionally.

0387 MECHANISM OF ACTION OF FUNDOPLICATION. *(E.)* Siewert, E.; Jennewein, H. M.; Waldeck, F. (no affiliation). *Proc. Int. Symp. Gastrointest. Motility, 4th, 1973*, p. 145-150.

Contractility studies were conducted on lower esophageal sphincters and on isolated muscle strips from various areas of the gastric wall of dogs and were compared to results of comparable studies on the reconstructed lower esophageal sphincter of humans following fundoplication. The function of the fundus cuff was investigated in 2 groups of 6 dogs each. In the first group, which had a normal sphincter but underwent fundoplication, the resting pressure of the esophageal sphincter and of the fundus cuff added up to an unphysiologically high pressure (45.5 mm Hg) compared to that of unoperated controls (20.0 mm Hg). The lower esophageal sphincter pressure almost doubled (81.0 mm Hg) following i.v. infusion of pentagastrin (3.0 µg/kg), a response proportionally the same as that of controls (49.3 mm Hg). Glucagon infusion (30 µg/kg) proportionally reduced lower esophageal sphincter tone in both controls (5.3 mm Hg) and fundoplicated animals (11.0 mm Hg), suggesting relaxation of both the fundus cuff and the sphincter. Elimination of the sphincter by circular myomectomy abolished sphincter tone (3.6 mm Hg) as well as the response to pentagastrin (4.0 mm Hg) and glucagon (1.0 mm Hg). Fundoplication following myomectomy restored both resting pressure (13.6) and response to pentagastrin (33.3 mm Hg) and glucagon (10.5) infusion. The results were correlated with sphincter pressures in human patients following fundoplication. Average resting pressure in such patients (17.3 mm Hg) compared favorably to those of unoperated controls without incompetent sphincters. The sensitivity to pentagastrin stimulation was somewhat reduced, with only a 12.7 mm Hg pressure increase. It was thus possible to demonstrate pentagastrin stimulation of the fundus cuff in humans. The therapeutic mechanism of fundoplication is therefore characterized by an increase in the sphincter pressure to higher levels which ensure gastroesophageal competence.

0388 PEDUNCULATED TUMOURS OF THE OESOPHAGUS. TWO CASES OF LIPOMA. *(E.)* Liliequist, B.; Wiberg, A. (Dep. Neuroradiol., Umea Univ., Sweden). *Acta Radiol. [Diagn.] (Stockh.)* 15(4):383-392, 1974.

0389 OUR EXPERIENCE WITH THE COMBINED THERAPY OF ESOPHAGEAL CANCER: TELEGAMMATHERAPY FOLLOWED BY SURGERY. *(Rus.)* Kochegarov, A. A.; Abdurasulov, D. M.; Vorobeva, L. I.; Kasymkhodzhaev, E. S.; Chernyshov, V. K. (Uabek Sci. Res. Inst. Oncol. Radiol., Tashkent, USSR). *Khirurgiia (Mosk.)* (2):86-93, 1974.

0390 SYMPTOMS OF GASTRO-ESOPHAGEAL REFLUX. *(Ger.)* Siegrist, P. W.; Krejs, G. J.; Blum, A. L. (Triemli Munic. Hosp., Zurich, Switzerland). *Dtsch. Med. Wochenschr.* 99(42):2088-2094, 1974.

0391 HEARTBURN AND GASTROESOPHAGEAL REFLUX. *(Ger.)* Krejs, G. J.; Brandli, H. H.; Seefeld, U.; Caro, G.; Blum, A. L. (Triemli Munic. Hosp., Zurich, Switzerland). *Helv. Med. Acta* 37(5/6): 405, 1974.

0392 BLEEDING ESOPHAGEAL VARICES. *(Ger.)* Ungeheuer, E. (Nordwest Hosp., Frankfurt/Main-Praunheim, Germany). *Langenbecks Arch. Chir.* 337:519-526, 1974.

0393 SCHATZKI-GARY LOWER ESOPHAGEAL RING IN SCLERODERMA. *(Sp.)* Pajares Garcia, J. M.; Rodriguez Cortes, J.; Martinez Pineiro, M.; Torralba, J. (Gran Hosp. del Estado, Madrid, Spain). *Rev. Esp. Enferm. Apar. Dig.* 44(2):167-174, 1974.

0394 PATHOLOGY OF THE ARTIFICIAL ESOPHAGUS MADE FROM SMALL INTESTINE AS SHOWN BY X-RAYS. *(Rus.)* Rozenfeld, L. G.; Bagirov, M. M. (Kiev Sci. Res. Inst. Otolaryngol., USSR). *Khirurgiia (Mosk.)* (2):93-98, 1974.

See also, 0331, 0405, 0431, 0673, 0718, 0748, 0749.

0395 INCIDENCE OF CARCINOMA OF STOMACH AND TUMOR TYPE. *(E.)* Whitehead, R.; Skinner, J. M.; Heenan, P. J. (Radcliffe Infirm., Oxford, England). *Br. J. Cancer* 30(4):370-372, 1974.

Gastrectomy specimens of 101 cases of carcinoma of the stomach recorded between 1940 and 1946 at a specific hospital in England were reviewed. A group of 22 cases of carcinoma of the stomach reported during 1971 and 1973 at the same hospital were also studied. The 2 groups of patients, separated by a period of 25 yr, were compared to determine if any changes in the type of malignancy (intestinal or diffuse), or in the precise location of atrophic gastritis and intestinal metaplasia in relation to the types of cancer could be noted. In both periods of time, the male to female ratio was about 2:1. The age range of the patients of each sex was not significantly different, nor was the population from which the 2 groups were drawn. There is no evidence that the methods of diagnosis or the criteria of selection for surgery have altered during the 25 yr intervening period. The ratios of intestinal to diffuse carcinoma were not significantly different. Atrophic gastritis and intestinal metaplasia had the same incidence in both types of cancer, and the distribtuion of these 2 lesions did not change during the 25 yr period. It is suggested that if the 2 main tumor types have a different etiological background, this is affected equally by whatever has been responsible for the falling rate of carcinoma of the stomach in England. Evidence suggests that environmental rather than racial or hereditary factors are responsible for the differences in the incidence of gastric cancer. However, in low risk areas, non-environmental factors must play a greater role in determining the cancer rate.

0396 HISTOPATHOLOGICAL STUDY ON DOUBLE CANCER OF THE STOMACH WITH SPECIAL REFERENCE TO COMBINATIONS OF HISTOLOGICAL TYPES. *(Jpn.)* Baba, Y.; Kakamura, K.; Sugano, H.; Takagi, K.; Nishi, M.; Kamukura, K. (Cancer Inst., Tokyo, Japan). *Gan No Rinsho* 19(1):28-38, 1973.

Out of 2408 cases of carcinoma of the stomach, resected from patients during the past twelve years, 153 cases showed multiple carcinoma. Of these, 110 cases of double carcinoma were studied in relation to combination of histological type, sex, age and cancer size. Classification of histological type was made if two carcinomas in the same stomach were of differentiated (D or d) or undifferentiated type (U or u; the capital letter represents a large carcinoma and the small letter, a small carcinoma). Seventy-five cases of double carcinoma were the combination of Dd; then, 22 cases of Ud, 8 of Uu, and 5 of Du, indicating that the smaller carcinomas tend to be of the undifferentiated type. The frequency of occurrence of multiple carcinoma was 6 times greater in males than in females. The Dd type double carcinoma in male patients was found five times more frequently than that in females; however, the ratio of occurrence of the Uu type of carcinoma by male and female was only 1.6. The average age of double carcinoma patients was 58.8, which was slightly higher than the average age of the 2408 stomach cancer patients, namely, 55.3. There was 10 years' difference between the average ages of patients with Dd and those with Uu type carcinomas. With increased patient age, more intestinal carcinoma was found, and also the differentiated type was found more often in the small carcinoma. With regard to carcinoma size, it was assumed that the size of a carcinoma is linearly related to its age. Small carcinoma developed independently of the size of large carcinoma. Carcinomas of both sizes tend to meet together during growth, under which circumstances the diagnosis of double carcinoma would be impossible. When the large carcinoma is of the differentiated type, the developing small carcinoma also is of the differentiated type, no matter how old the large carcinoma is. However, when the large carcinoma is of the undifferentiated type, the small carcinoma tends to be of the undifferentiated type provided the large carcinoma is a relatively young one, but the small carcinoma tends to be of the differentiated type when the large carcinoma is an old one.

0397 A POPULATION SCREENING FOR FETAL SULFOGLYCOPROTEIN ANTIGEN IN GASTRIC JUICE. *(E.)* Hakkinen, I. P. T. (Dep. Pathol., Univ. Turku, Finland). *Cancer Res.* 34(11):3069-3072, 1974.

The results of screening for fetal sulfoglycoprotein antigen in the age group of 40-65 are presented. Of the three populations considered, 2 were composed of industrial personnel and 1 was composed of unselected rural people. A total of about 14,000 people participated. Fetal sulfoglycoprotein antigen was present in the gastric juice in about 3% of the industrial and from 5-7% in the rural population. Three histologically verified cases and 1 suspected case of gastric cancer, all clinically symptomless, were found among the fetal sulfoglycoprotein antigen-positive cases. In addition, there was 1 case of gastric polyposis. All of these cases underwent radical surgery. In addition, benign alterations were found in the examination of 461 fetal sulfoglycoprotein antigen positives. These included benign ulcer in 10 cases, deformation of pylorus in 12, erosion in 8, resection due to earlier ulcer in 15, gastritis in 61, atrophy in 23, and polyps in 15. These alterations, 144 in total, were single or combined in 126 persons. The follow-up since 1970 had shown no new case of clinical gastric cancer appearing among the fetal sulfoglycoprotein antigen-negative persons. The possibility of using this screening method may be significant in early cancer detection.

0398 MUCOSAL GLYCOPROTEINS IN CARCINOMA CELLS OF GASTROINTESTINAL TRACT, AS DETECTED BY IMMUNOFLUORESCENCE TECHNIQUE. *(E.)* Kawasaki, H.; Kimoto, E. (Kurume Univ. Sch. Med., Japan). *Acta Pathol. Jpn.* 24(4):481-494, 1974.

Antisera, obtained by immunization of human or porcine gastric mucosa-specific (GMP) and intestinal mucosa-specific glycoprotein (IMP) into rabbits, were employed for the immunofluorescence staining of

carcinoma cells in the gastrointestinal tract. In normal mucosa, the fluorescence for GMP was seen in the PAS-alcian blue-stainable mucous cells of gastric glands. The intensity of the fluorescence correlated well with the distribution of PAS stain. The fluorescence for GMP was scarcely demonstrable in any intestinal mucosa cells except Brunne's cells of the duodenum. Fluorescence for IMP was scarcely demonstrable in the gastric mucosa. The alcianophilic goblet cells in the intestinal mucosa were intensely fluorescent with anti-IMP antiserum. Immunohistological studies on carcinoma cells of the gastrointestinal tract showed that the well-differentiated carcinoma cells of adenocarcinoma type, having an orderly arranged glandular pattern, were devoid of fluorescence. The diffusely and dissociated proliferating carcinoma cells fluoresced well for GMP and IMP. Carcinoma cells of less-differentiated and less orderly arranged glandular structures were also fluorescent. In general, the intensity of fluorescence correlated well with the disordering of glandular structure. The distribution of fluorescence of GMP correlated well with the PAS-positive neutral mucus, while that for IMP distributed concordantly with the acid mucus of PAS-alcian blue-positive staining. The gastric mucosal glycoprotein appeared in most cases of rectal carcinomas in association with the intestinal one, indicating that gastric metaplasia occurred in rectal carcinoma in the same way as intestinal metaplasia did in gastric carcinoma. Carcinoma cells metastasizing into serous effusion singly or in small clusters fluoresced well. Malignant cells in effusions, metastasizing from other organs, were not fluorescent. Any cells in effusion of non-cancerous disease, i.e. cirrhosis were scarcely fluorescent. Thus, this immunofluorescence technique is valuable in distinguishing the primary lesion of malignant cells in effusions. If a causal relation exists between the deletion of normal antigens and carcinogenesis, these findings of similar patterns of antigenic modification suggest some common link in the carcinogenic process. Uncontrolled growth could be due to loss of certain cellular constituents that enable normal cells to respond to regulative stimuli.

0399 GEOGRAPHICAL PATHOLOGY OF GASTRIC CARCINOMA. *(E.)* Kubo, T. (Fac. Med., Kyushu Univ., Fukuoka, Japan). *Acta Pathol. Jpn.* 24(4):465-479, 1974.

An international comparative study was made of gastric carcinoma in order to determine whether the tumor showed different morphological features in different countries. The series were from Japan, Korea, USA, New Zealand, and Chile. Further comparison was made at different time periods in the USA, New Zealand, and Japan. The majority of tumors were found in the lower 2/3 of the stomach in all countries with no marked international difference. In all countries the predominant type among older patients was the adeno-mucoid type; in younger patients, the diffuse type. Therefore, in countries with a larger number of younger patients, an apparently higher incidence of the diffuse type was noted. After compensating for the difference in patient populations, a slight difference in the number of both types (diffuse and adeno-mucoid) was noted in different countries, but this showed no uniform trend in regard to the different mortality rates in the countries. Within Japan there were differences in mortality rates for various areas of the country. However, the data showed that there was no geographical difference with regard to histological type whether mortality rates were high or low. In a study of data from the USA, where mortality rates due to gastric carcinoma are declining, no change in histological type could be linked with this decline. It was concluded that there is no significant difference in histological appearance of gastric carcinoma either in different countries or at different times. It is suggested that a common factor may be operating throughout the world as a gastric carcinogen.

0400 INDOLENT MUCOID CARCINOMA OF STOMACH. *(E.)* Brander, W. L.; Needham, P. R. G.; Morgan, A. D. (Westminster Med. Sch., London, England). *J. Clin. Pathol.* 27(7):536-541, 1974.

Histological sections of 574 cases of carcinoma of the stomach, either biopsied (17 cases) or resected (557 cases) were reviewed. If 50% or more of the bulk of the tumor represented in the sections was epithelial mucus, either intracellular or extracellular, the carcinoma was classified in the mucoid group. Of the 574 carcinomas, 50 (8.7%) were classified as mucoid. Of these, 41 (24 men and 17 women, aged 37-83 yr) were of mixed pattern, 3 (all men, aged 51-80 yr) were of pure signet-ring type, and 6 (all men, aged 45-76 yr) were easily recognizable as the well differentiated type. Treatment in 49 of these cases was by partial or total gastrectomy; 1 of the signet-ring carcinomas was inoperable. Survival of those with mucoid carconoma of mixed pattern was an average of 18 months. Even excluding those in whom distant metastases was noted at the time of operation, this period could only be extended to 21 months. The 3 cases of signet-ring pattern had a mean survival time of 5 months. In contrast, the 6 cases with well differentiated mucoid carcinoma had a mean survival time of 9 yr, even including those with lymph-node involvement. Preoperative history in all cases ranged from 4 months to several years and was not significantly different for any of the 3 types. Thus the accepted view that mucoid carcinoma of the stomach has a poor prognosis may not hold for those patients with the well differentiated type.

0401 NATURAL HISTORY OF AUTOIMMUNE ACHLORHYDRIC ATROPHIC GASTRITIS. A 1-15 YEAR FOLLOW-UP STUDY. *(E.)* Irvine, W. J.; Cullen, D. R.; Mawhinney, H. (R. Infirm., Edinburgh, Scotland). *Lancet* (7819):482-485, 1974.

A series of 90 patients (aged 23-77 yr) with achlorhydric autoimmune atrophic gastritis has been followed from 1-15 yr (mean 6 yr). During this time a total of 17 patients (19%) developed malabsorption of vitamin B_{12}. The interval between the diagnosis of atrophic gastritis and the development of vitamin

B_{12} malabsorption varied from 1-15 yr. Of these 90 patients, 20 (22%) had serum-type-I intrinsic-factor antibody; and, of these 20, 7 (35%) developed vitamin B_{12} malabsorption during follow-up. However, an appreciable number of patients without serum type-I intrinsic-factor antibody also developed vitamin B_{12} malabsorption. Serial serum intrinsic-factor antibody titers were of no value in predicting those patients liable to develop vitamin B_{12} malabsorption. These findings strongly suggest that factors other than the presence or titer of serum antibody to intrinsic factor, or to gastric parietal-cell microsomal antigen, are involved in the progression of achlorhydric atrophic gastritis to vitamin B_{12} malabsorption. In conclusion, this study demonstrates the slow but steady progression of achlorhydric atrophic gastritis to pernicious anemia in a group of patients presenting with endocrine disorders.

0402 ATROPHIC GASTRITIS AND ITS SEQUELAE. RESULTS OF 19-23 YEARS' FOLLOW-UP EXAMINATIONS. *(E.)* Siurala, M.; Lehtola, J.; Ihamaki, T. (Meilahti Hosp., Helsinki, Finland). *Scand. J. Gastroenterol.* 9(5):441-446, 1974.

Follow-up studies were performed on 116 subjects with atrophic gastritis. The mean follow-up time was 21 yr and the mean age of the patients at re-examination was 64 yr. Of the original 116 subjects, 34 died, leaving 82 patients to be studied during this last re-examination. Of these 82, 28 patients died during the follow-up period due to: gastric carcinoma (2); colonic cancer (1); pulmonary cancer (2); ovarian cancer (1); cerebral tumor (1); abdominal cancer (1); cardiovascular disease (15); and other diseases (5). One of the patients who died of gastric carcinoma had been so diagnosed during the previous follow-up period 8 yr prior to death. Of the remaining 54 patients, re-examinations were performed on 35. Gastroscopy disclosed polypoid lesions in 6 cases (true adenomas in 2, hyperplasias in 3, and inflammatory polyp in 1). Multiple direct vision gastric biopsy from the antrum and the body revealed that gastritic changes were somewhat more pronounced in the body than in the antrum. Moderate or severe epithelial atypia was present in 13 of the 35 biopsied cases. Parietal cell antibodies were found in 3 and intrinsic factor antibodies in an additional 2 cases. Serum gastrin values were above 75 pM/liter in 24 cases and in 1 out of 24 controls. Serum vitamin B_{12} levels were within normal limits in all cases. Many patients suffered abdominal discomfort. Epigastric complaint or difficulty associated with food intake was present in 18. There were 14 patients who appeared essentially symptomless with regard to abdominal organs. There was no correlation between the subjective complaints and the morphological state of the antral or body mucosa. Although atrophic gastritis preceded the occurrence of gastric tumors in all cases diagnosed during the time of observation, sources of error inherent in the present follow-up method disallow the conclusion that atrophic gastritis is in general a precancerous condition.

0403 ASPIRIN INDUCED GASTRIC MUCOSAL INJURY IN RATS AND ADENYL CYCLASE. *(E.)* Mangla, J. C.; Kim, Y. M.; Rubulis, A. A. (Monroe Community Hosp., Rochester, N.Y.). *Biochem. Med.* 11(4):376-382, 1974.

The effect of aspirin on gastric mucosal adenyl cyclase in male Holtzman rats was studied. Rats were given aspirin (150-300 mg/kg, by stomach tube) after an overnight fast. The mucosa of the stomach was examined after a given time ranging from 5 min to 5 hr. The mucosa appeared congested, red, hemorrhagic with many erosions. The effect of aspirin became apparent as early as 5 min after the oral dose with maximal adenyl cyclase stimulation noted at 4 hr. For *in vitro* studies, gastric mucosal homogenate was treated with aspirin dissolved in methyl cellulose at concentrations of 0.3, 0.6, 0.9, and 1.8 mm/0.38 ml of reaction mixture. Again adenyl cyclase stimulation was observed and a direct correlation between aspirin concentration and the degree of adenyl cyclase activity was noted. Adenyl cyclase activity increased linearly with aspirin concentration from 0.3 mg up to 1.8 mg in .38 ml reaction mixture. The stimulation of adenyl cyclase by aspirin may exhaust the ATP stores quickly, causing mucosal injury.

0404 THE PROBLEM OF "EARLY GASTRIC CANCER". *(Sp.)* Herrerias, J. M.; Aljama, P.; Jimenez Pereperez, J. A.; Damiano, A.; Osorio, M.; Nararro y Esteban, F. (Fac. Med. Univ. Sevilla, Cadiz, Spain). *Rev. Esp. Enferm. Apar. Dig.* 44(2): 157-166, 1974.

0405 COMPARISONS BETWEEN NEUTRON AND TELECOBALT THERAPIES IN BRONCHIAL, GASTRIC, AND ESOPHAGEAL CARCINOMAS. *(Ger.)* Eichhorn, H. J.; Lessel, A.; Matschke, S. (Cent. Inst. Cancer Res., Berlin-Buch, Germany). *Strahlentherapie* 147(6):559-563, 1974.

0406 CARCINOMA IN THE GASTRIC STUMP FOLLOWING RESECTION FOR BENIGN DISEASE: A CRITICAL EVALUATION. *(Ger.)* Peters, H.; Schubert, H. J.; Reifferscheid, M. (Med. Fac., Rhenish Westphalian Tech. Coll., Aachen, Germany). *Langenbecks Arch. Chir.* 336(3):219-233, 1974.

0407 MENETRIER'S DISEASE WITH MALIGNANT DEGENERATION TREATED BY TOTAL GASTRECTOMY. *(Fr.)* Braillon, G.; Boulanger, J. P. (Jules Courmont Hosp., Pierre-Benite, France). *Lyon Chir.* 70(5): 326-327, 1974.

0408 THE RELATIONSHIP BETWEEN CHRONIC GASTRITIS, GASTRIC ULCERATION AND CARCINOMA OF THE STOMACH. A HISTORICAL REVIEW. *(E.)* Bock, O. A. A. (Tygerberg Hosp., Tiervlei, South Africa). *S. Afr. Med. J.* 48(49):2063-2066, 1974.

0409 CHRONIC GASTRITIS IN THE MONGOLIAN PEOPLE'S REPUBLIC. *(Ger.)* Wolff, G.; Badral, Z.; Jamin-Scharav (Cent. Inst. Cancer Res., Berlin, Germany). *Dtsch. Z. Verdau. Stoffwechselkr.* 33(1): 37-39, 1974.

0410 TREATMENT OF HEMORRHAGIC GASTRITIS BY THE ENDOSCOPIC APPLICATION OF ACRYLIC POLYMER. *(E.)* Keller, R. T.; Logan, G. M. (Univ. North Dakota Sch. Med., Grand Forks). *Gastrointest. Endosc.* 21(2):75-76, 1974.

0411 ENDOSCOPIC DIAGNOSIS OF GASTRIC EROSIONS AND THEIR CLINICAL SIGNIFICANCE. *(Ger.)* Bosseckert, H.; Koppe, P.; Heid, R. (Med. Clin., Friedrich Schiller Univ., Jena, Germany). *Z. Gesamte Inn. Med.* 29(21):888-891, 1974.

0412 POSTOPERATIVE AND STRESS BLEEDING. *(Ger.)* Farthmann, E.; Horatz, K. (Surg. Clin., Univ. Hamburg, Germany). *Langenbecks Arch. Chir.* 337:541-545, 1974.

0413 PEPTIC ULCERS WHICH DEVELOPED AND WERE CURED DURING UNINTERRUPTED CORTICOSTEROID THERAPY: 11 CASES. *(Fr.)* Etienne, J. P.; Labayle, D.; Chaput, J. C.; Buffet, C. (Antoine Beclere Hosp., Clamart, France). *Ann. Med. Interne (Paris)* 125(11): 823-829, 1974.

0414 METABOLIC CHANGES IN GASTRECTOMIZED PATIENTS. *(Por.)* Ribeiro, E. B. (Sao Lucas Sanit., Sao Paulo, Brazil). *Bol. Sanatorio Sao Lucas* 35(6):119-120, 1974.

0415 SOME ASPECTS OF CARBOHYDRATE METABOLISM IN PATIENTS WITH DUMPING SYNDROME. *(Pol.)* Lubczynska-Kowalska, W. (Acad. Med., Wroclaw, Poland). *Pol. Tyg. Lek.* 29(33):1447-1450, 1974.

0416 NEONATAL GASTRIC PERFORATION NOT CAUSED BY CONGENITAL MUSCULAR DEFECTS. *(E.)* Priebe, Jr., C. J.; Mangahas, T.; Garret, R. (Roosevelt Hosp., New York, N. Y.). *Arch. Pathol.* 98(6): 422-425, 1974.

0417 RELATIONSHIPS BETWEEN THE INDICES OF THE STOMACH ELECTRICAL ACTIVITY IN CERTAIN PATHOLOGICAL CASES. *(E.)* Altuparmakov, I. A.; Mizhorkova, Z. N. (Med. Acad. Sofia, Bulgaria). *Dokl. Acad. Nauk (Bulg.)* 27(7):999-1002, 1974.

0418 ISOLATED HODGKIN'S DISEASE OF THE STOMACH. *(Ger.)* Takacsi-Nagy, L. (Emil Weil Inst. Cancer Radiol., Budapest, Hungary). *Strahlentherapie* 147(5):477-481, 1974.

0419 ELECTRON MICROSCOPE STUDY OF HUMAN GASTRIC MUCOSA IN A CASE OF HEMOCHROMATOSIS. *(Pol.)* Domagala, W.; Marlicz, K. (Pomeranian Acad. Med. Szczecin, Poland). *Patol. Pol.* 25(1):153-158, 1974.

0420 ANTRUM MORPHOLOGY AND SERUM GASTRIN LEVEL IN ACHLORHYDRIA. *(Ger.)* Miederer, S. E.; Kutz, K.; Lindstaedt, H.; Stadelmann, O.; Vetter, H.; Wobser, E.; Elster, K. (Med. Polyclin., Univ. Bonn, Germany). *Dtsch. Med. Wochenschr.* 100(2):51-53, 1975.

See also, 0332, 0349, 0350, 0351, 0352, 0353, 0424, 0425, 0426, 0430, 0431, 0448, 0458, 0497, 0718, 0736, 0748, 0803.

STOMACH

Peptic Ulcers

0421 A FOLLOW-UP REPORT OF A PROSPECTIVE EVALUATION OF VAGOTOMY-PYLOROPLASTY AND VAGOTOMY-ANTRECTOMY FOR TREATMENT OF DUODENAL ULCER. *(E.)* Jordan, Jr., P. H. (Baylor Coll. Med., Houston, Tex.). *Ann. Surg.* 180(3):259-264, 1974.

Two evaluation studies were done on 200 patients treated for duodenal ulcer at 3-5 and 5-8 yr after surgery. Truncal vagotomy and drainage was performed on 108 (with 2% mortality) and 92 cases had truncal vagotomy and antrectomy (no mortality). A complete study was done on 94% of all patients, including 30 who died of unrelated causes before completion. Gastrointestinal symptoms were about equal for both groups when the 1st evaluation was done, but symptoms appeared to occur less in the vagotomy and drainage group at the time of the second evaluation. However, it was noted that a total of 9 patients in the vagotomy and drainage group had been reoperated for recurrent ulcer as opposed to only 1 patient in the vagotomy and antrectomy group. To assess completeness of vagotomy, insulin stimulation tests were performed on 183 patients 937 times (6 months-8 yr postsurgery). There were 42% and 86% negative results in the vagotomy and drainage and in the vagotomy and antrectomy, resp. The basal acid output and response to histalog stimulation were significantly reduced in both groups of patients, though greater in the vagotomy and antrectomy cases. It is concluded that the vagotomy and antrectomy procedure is superior in view of the fewer recurrences.

0422 THE PANCREATIC EXOCRINE SECRETION IN DUODENAL ULCER PATIENTS BEFORE AND AFTER SELECTIVE PROXIMAL VAGOTOMY OF THE STOMACH. *(E.)*

Berstad, A.; Roland, M.; Petersen, H.; Liavag, I. (Deaconess Hosp., Oslo, Norway). *Scand. J. Gastroenterol.* 9(5):431-436, 1974.

Pancreatic response to varying doses of exogenous secretin was examined in 11 duodenal ulcer patients (2 women and 9 men, aged 27-60 yr) before and after selective proximal vagotomy and in 12 healthy subjects. Gastric and duodenal secretions were collected during i.v. infusion of secretin in step-wise increasing doses of 0.08, 0.233, 0.7, and 2.1 CU/kg/hr. Bicarbonate output was lower at all doses in patients than in controls, significantly so for the 0.23 CU/kg/hr dose. The maximal response was the same in patients and controls (0.33 mEq/kg/30 min). The dose of secretin giving half-maximal response was 0.41 CU/kg/hr in controls and 0.62 CU/kg/hr in patients. Bicarbonate output increased, but not significantly after selective proximal vagotomy in response to the 3 lowest doses of secretin. The mean response to the highest dose of secretin decreased significantly from 17.3 to 15.6 mEq/30 min after operation. The maximal response of 0.26 mEq/kg/30 min after vagotomy was not significantly different than that before operation. The dose of secretin giving half-maximal response after vagotomy was 0.35 CU/kg/hr, significantly lower than before, but not significantly different from control values. The output of proteolytic enzymes in response to the 2 lowest doses of secretin before operation was significantly higher in patients than in controls. After operation the enzyme output was higher following administration of the 2 highest doses, significantly so at the 0.7 CU/kg/hr level. In duodenal ulcer patients the enzyme output before vagotomy remained at the same level following the 3 lowest doses of secretin and increased significantly with the highest dose. After operation, the curve started rising significantly with the second highest dose. These results suggest a lower sensitivity of the bicarbonate-secreting cells of the pancreas in duodenal ulcer patients, and that the sensitivity is increased by selective proximal vagotomy. Also, for enzyme secretion, the operation apparently shifts the dose-response curve to the left. There may be a greater leakage of bile into the duodenum in duodenal ulcer patients, even more so after selective vagotomy, than in normal persons.

0423 PREVENTION OF RESTRAINT AND INDOMETHACIN-INDUCED GASTRIC ULCERATION BY BILE DUCT OR PYLORUS LIGATION IN RATS. *(E.)* Hemmati, M.; Abtahi, F.; Farrokhsiar, M.; Djahanguiri, B. (Sch. Med., Univ. Tehran, Iran). *Digestion* 10:108-112, 1974.

The influence of bile duct and pylorus ligation was investigated on two experimental models of gastric ulceration: restraint- and indomethacin-induced gastric mucosal ulcerations in rats of both sexes. Animals were divided into 6 groups of 40 rats as follows: (1) restraint; (2) restraint + bile administration; (3) indomethacin (40 mg/kg i.p.); (4) indomethacin + bile; (5) bile alone; and (6) saline. In bile duct-ligated rats, 10% of the restrained and none of the indomethacin treated animals showed gastric ulceration. These figures were 10% and 50% resp. in pylorus-ligated rats. Administration of bile to restrained and indomethacin-treated rats nullified the preventive effect of bile duct or pylorus ligation, suggesting that the preventive effect was due to the lack of bile reflux into the stomach. However, the possible role of bile duct ligation or pylorus ligation in reducing gastric secretion of acid should not be disregarded. The failure of pylorus ligation to completely prevent indomethacin-induced ulcerations may be due to a direct effect of the drug on the stomach. Bile salts have been shown to be cytolytic, to damage the gastric mucosa, to release gastrin, and to cause gastric ulceration; but the precise mechanism whereby the presence of bile in the stomach promotes the pathogenesis of gastric ulceration following stress or indomethacin is not clear.

0424 EXPERIMENTAL STRESS ULCERS IN ACIDOTIC AND NONACIDOTIC RENAL INSUFFICIENCY. *(E.)* Fischer, R. P.; Peter, E. T.; Mullane, J. F. (Albuquerque VA Hosp., N.M.). *Arch. Surg.* 109(3): 409-411, 1974.

The study of stress ulcer pathogenesis (with the restraint method of Brodie and Hanson used as the model) was performed on groups of rats which exibited acidotic and non-acidotic renal insufficiency. Two experiments several months apart were performed on control and experimental Sprague-Dawley rats (200-225 g). For 24 hr prior to restraint the rats received water but no food. They were slightly anesthetized with ether and restrained. In one experiment the rats were restrained for 11 hr and in another experiment, for 24 hr. Acidotic renal insufficiency was induced in the experimental group by a single i.p. injection of 4 mg/100 ml uranyl nitrate. Controls received saline (0.5 ml/100 g, i.p.). Four days after injection the rats were restrained. The uranyl rats were severely acidotic (mean pH 7.19) and hyperkalemic (mean potassium concentration 5.4 mEq/liter). Erosions (3 or more) were present in 12 experimental and 3 control rats. One or more ulcers were found in 10 experimental and 3 control rats. Acidotic hyperkalemic renal insufficiency thus enhances restraint-induced stress ulcers. Non-acidotic renal insufficiency was induced in rats by partial nephrectomy performed on the experimental rats on the 1st day, while on the same day the controls underwent sham flank operations. Eight days later, contralateral nephrectomies were done on both the experimental and control rats. All were restrained on the 14th day. Blood urea nitrogen (BUN) levels were slightly if at all elevated in the controls after contralateral nephrectomy and only a few were significantly uremic by the day of restraint. One or more gastric erosions were present in 12 experimental and 4 control rats; 1 or more ulcers were found in 5 experimentals and 1 control rat. These rats were not acidotic nor hyperkalemic, but did have moderate uremia (average BUN level 111.6 mg/100 ml) and it appeared that this enhanced the restraint-induced stress erosion and ulcer formation in this group. In a separate set of experiments, both groups were duplicated but the rats were killed without restraint. The latter had no gastric ulcers and only an average of 0.17 erosions/rat. It is con-

cluded that renal insufficiency interferes with normal gastric mucosal repair and that erosive gastritis is an integral component of uremic enterocolitis found in severely uremic patients.

0425 HYPERGLUCAGONEMIA, HYPOCALCEMIA AND DIMINISHED GASTRIC BLOOD FLOW: EVIDENCE FOR AN ETIOLOGICAL ROLE IN STRESS ULCER OF RAT. *(E.)* Schwille, P. O.; Schellerer, W.; Reitzenstein, M.; Hermanek, P. (Dep. Surg., Univ. Erlangen-Nuremberg, Erlangen, Germany). *Experientia* 30(7):824-826, 1974.

Three groups of male Wistar rats of 200 g each were randomly divided into intact, sham operated (laparotomy only) and adrenalectomized animals. One moiety served as control and the other half was stressed by restraint for 24 hr. Food and water were withheld for 24 hr prior to experiment (controls). Following stress, all animals were anesthetized and laparotomized to expose the gastric surface and allow bleeding from the aorta. Oxygen pressure determination was performed by polarography. Radioimmunoassay measured the values of pancreatic glucagon and gastrin. Serum calcium was determined by EDTA titration. The ulcer index (UI) according to Brodie and Hanson served as a criterion for gastric lesions induced by stress. Intact and sham-operated rats had zero UI and nearly equal oxygen pressure values (19.08 *versus* 18.58). Both stress alone and adrenalectomy alone severely increased the UI and reduced gastric perfusion, the lowest oxygen pressure being found in adrenalectomized rats following stress. Pancreatic glucagon in stressed rats was definitely elevated (corroborating the view that catecholamines are potent stimuli to α-cells). Immunoassayable gastrin tended to decline during stress but increased markedly after adrenalectomy. Serum calcium was diminished in stressed animals with the exception of those who had undergone adrenalectomy. It is suggested that gastric ulcers originally induced by stress situations can be characterized by a triad of hyperglucagonemia, hypocalcemia and reduced gastric mucosa blood supply.

0426 STARVATION, GLUCOSE, AND STRESS ULCERS IN THE RAT. *(E.)* Mullane, J. F.; Pyant, Jr., R. L.; Wilfong, R. C.; Dailey, W. (Walter Reed Army Inst. Res., Washington, D.C.). *Arch. Surg.* 109(3): 416-419, 1974.

Male Wistar rats (35-120 g) were divided into several groups with similar mean weights. The control group had free access to food and water until 20 hr prior to restraint, when food was removed. The experimentally starved rats received only water for 96 hr before restraint. During this 96 hr period the rest of the groups received 1.0, 2.5 and 5.0 g/100 ml concentrations of glucose in their drinking water but no food. The control rats received 39 calories/day; the group receiving 5.0 g had 13 calories. All rats were restrained for a period of 7 hr; they were then sacrificed and their stomachs removed and examined. The evaluation was done for two groups: one consisted of controls, starvation and 5.0 g/100 ml of glucose ingestion; the other group was composed of controls, 1.0, 2.5 and 5.0 g/100 ml glucose ingestion. The starved rats had the largest number of restraint induced gastric erosions and were closely followed by the groups that received 1.0 and 2.5 g/100 ml of glucose. The erosions found in the group that received 5.0 g/100 ml were about the same in number as in the control group. It appears that a critical degree of caloric intake does protect the gastric mucosa from the effects of stress.

0427 DIAGNOSIS OF PYLORODUODENAL ULCERS BY RADIOLOGY AND ENDOSCOPY. *(Fr.)* Realini, S.; Bugnion, M.; Fluckiger, A.; Hofstetter, J. R. (Med. Polyclin., Univ. Lausanne, Switzerland). *Schweiz. Med. Wochenschr.* 105(1):19-20, 1975.

0428 OTHER PERSPECTIVES FOR EXPLAINING THE PATHOGENESIS OF PEPTIC ULCER. *(Por.)* Ribeiro, E. B. (Sao Lucas Sanit., Sao Paulo, Brazil). *Bol. Sanit. Sao Lucas* 36(2):45-48, 1974.

0429 DEGLYCYRRHIZINISED LIQUORICE: A REPORT OF ITS PHARMACOLOGICAL PROPERTIES AND THERAPEUTIC EFFICACY IN PEPTIC ULCER. *(E.)* Brogden, R. N.; Speight, T. M.; Avery, G. S. (Australasian Drug Inf. Serv., Auckland, New Zealand). *Drugs* 8(5): 330-339, 1974.

0430 CURATIVE ACTIVITY OF A SULPHATED POLYGALACTOSIDE (CP1200) ON EXPERIMENTAL GASTRIC ULCERS IN THE RAT. *(E.)* Lambelin, G.; Roba, J. (Res. Lab., Machelen, Belgium). *Arch. Int. Pharmacodyn.* 211(1):18-23, 1974.

0431 EXPERIENCE WITH METOCLOPRAMIDE IN GASTROINTESTINAL DISEASE: CLINICAL STUDY AND INVESTIGATION OF SIDE EFFECTS IN 1000 ORIGINAL CASES. *(Sp.)* Fornes Pons, J. (no affil.). *Rev. Esp. Enferm. Apar. Dig.* 44(4):421-428, 1974.

0432 MEDICAL AND SURGICAL THERAPY FOR PEPTIC ULCER. *(It.)* Pezzuoli, G.; Spina, G. (Inst. Surg. Pathol., Univ. Padua, Italy). *Clin. Eur.* 12(5):431-439, 1974.

0433 HIGHLY SELECTIVE VAGOTOMY. *(E.)* Johnston, D. (Gen. Infirm., Leeds, England). *Gut* 15(9):748-757, 1974.

0434 PROXIMAL GASTRIC VAGOTOMY. *(E.)* Wastell, C.; Wilson, T.; Pigott, H. (Gordon Hosp., London, England). *Proc. R. Soc. Med.* 67(11):1183-1185, 1974.

0435 ACCIDENTS AND COMPLICATIONS IN SELECTIVE AND PROXIMAL GASTRIC VAGOTOMY. *(E.)* Kalaja, E.; Clemmesen, I.; Banke, L.; Kragelund, E.; Christiansen, P. M. (Bispebjerg Hosp., Copenhagen, Denmark). *Surgery* 77(1):140-143, 1975.

0436 PYLORUS-PRESERVING GASTRECTOMY FOR GASTRIC ULCER—ONE- TO NINE-YEAR FOLLOW-UP STUDY. *(E.)* Sekine, T.; Sato, T.; Maki, T.; Shiratori, T. (Tohoku Univ. Sch. Med., Sendai, Japan). *Surgery* 77(1):92-99, 1975.

0437 ULCERS IN THE UPPER POLE OF THE STOMACH: SURGICAL PROBLEMS. *(Fr.)* Edelmann, G.; Boutelier, P.; Champault, G. (Bichat Hosp., Paris, France). *Lyon Chir.* 70(5):299-302, 1974.

0438 COMMON MESENTERY DIAGNOSED DURING SURGICAL TREATMENT OF A STOMACH ULCER. *(Fr.)* Loup, J. (Hosp. Cent., Auch, France). *Lyon Chir.* 70(5): 325-326, 1974.

0439 THE EFFECT OF KNOWN COMPLETE HEALING ON THE NATURAL HISTORY OF CHRONIC GASTRIC ULCER. *(E.)* Greig, M.; Hobbin, E.; Hunt, J.; Coupland, G. A. E.; Piper, D. W. (R. North Shore Hosp., Sydney, Australia). *Aust. NZ J. Med.* 4(3):302, 1974.

0440 FISTULAS BETWEEN THE GALLBLADDER, COMMON BILE DUCT, AND DUODENUM AS A COMPLICATION OF CHRONIC DUODENAL ULCER. *(Ger.)* Haas, D.; Vogt, B. (Cantonal Hosp., Lucerne, Switzerland). *Helv. Chir. Acta* 41(5/6):799-801, 1974.

0441 MASSIVE HEMORRHAGE FROM GASTRIC AND DUODENAL ULCERS: EMERGENCY THERAPY. *(Ger.)* Eichfuss, H. P.; Soehendra, N.; Wehling, D. (Surg. Clin., Univ. Hamburg, Germany). *Dtsch. Med. Wochenschr.* 99(35):1732-1734, 1974.

0442 GASTROPLEGIA SECONDARY TO VAGOTOMY. *(Fr.)* Larcan, A.; Laprevote-Heully, M. C.; Lambert, H.; Fieve, G.; Fresse, A. (Univ. Hosp. Cent., Nancy, France). *Arch. Fr. Mal. App. Dig.* 63(4):315-318, 1974.

0443 MORPHOLOGICAL FEATURES AND ABSORPTIVE FUNCTION OF MUCOSA IN THE EMPTY INTESTINE AFTER PARTIAL GASTRECTOMY FOR PEPTIC ULCER. *(Pol.)* Szklanny, J. (Acad. Med. Lodz, Poland). *Pol. Tyg. Lek.* 30(40):1715-1718, 1974.

0444 FUNCTIONAL AND HISTOMORPHOLOGICAL CHANGES IN THE LIVER IN PEPTIC ULCER. *(Ger.)* Temelkov, S.; Badakov, S. (I. P. Pavlov Higher Med. Inst., Plovdiv, Bulgaria). *Folia Med. (Plovdiv)* 15(4):207-214, 1973.

0445 CYTOCHEMICAL CHANGES OF ENTEROCHROMAFFINE-LIKE [SIC!] CELL IN THE EXPERIMENTAL GASTRIC ULCERS. *(E.)* Mori, J.; Fujiwara, M. (Fujisawa Res. Lab., Osaka, Japan). *Acta. Histochem. Cytochem.* 7(1):79-80, 1974.

0446 CHANGES IN THE SERUM ALKALINE PHOSPHATASE ACTIVITY AND SERUM PROTEINS IN ULCER PATIENTS BEFORE AND AFTER SURGERY. *(Ger.)* Temelkov, S.; Taralov, S. (I. P. Pavlov Higher Med. Inst., Plovdiv, Bulgaria). *Folia Med. (Plovdiv)* 15(5):257-264, 1973.

0447 BLOOD AMMONIA DETERMINATIONS IN GASTRECTOMIZED PATIENTS. *(Ger.)* Schwamberger, K.; Falser, N. (Surg. Clin., Univ. Innsbruck, Austria). *Langenbecks Arch. Chir.* 335(4):351-357, 1974.

0448 CURRENT POSSIBILITIES OF RESEARCH AND SIGNIFICANCE OF THE GASTRIC MUCOSAL BARRIER. *(E.)* Barbara, L.; Miglioli, M.; Baldi, F.; Cornia, G. L.; Labo, G. (Inst. Pathol. Med. I, Univ. Bologna, Italy). *Rend. Gastroenterol.* 5(3):217-218, 1973.

See also, 0045, 0096, 0103, 0333, 0347, 0408, 0417, 0486, 0718, 0729.

SMALL INTESTINE

0449 MALIGNANCY, WEIGHT LOSS, AND THE SMALL INTESTINAL MUCOSA. *(E.)* Barry, R. E. (Bristol R. Infirm., England). *Gut* 15(7):562-570, 1974.

The mucosal architecture and dynamics of the small bowel were studied in 2 groups of patients. One group consisted of patients with malignant disease not of the gastrointestinal tract, but associated with severe weight loss (mean 21 lb). The 2nd group consisted of patients who had lost at least 28 lb for reasons other than malignant or gastrointestinal disease. Mucosal architecture in malignant disease was significantly different from normal. The presence of cylindrically shaped villi and more than 1 leaf-shaped villus were diminished in malignancy, while the incidence of ridges and convolutions increased in groups with malignancies or wasting illness of another type. The mean mucosal thickness in normal controls was 522 μ which was reduced to 443 μ in patients with malignant disease. Similar decreases were noted in patients with wasting diseases other than malignancy. The malignant group showed a significant decrease in the rate of lactose absorption. The epithelial cell (DNA) loss rate from intestinal mucosa was decreased in patients with malignant and other wasting diseases. As there must

be dynamic equilibrium between the rates of epithelial cell production by the crypts and their loss from the villi, then the epithelial cell turnover is likely to be reduced as well. This implies that the architectural changes observed in the jejunum of patients with malignant disease are caused by hypoplasia of the mucosa.

0450 PROTEIN-LOSING ENTEROPATHY IN PRIMARY LYMPHOEDEMA: MESENTERIC LYMPHOGRAPHY AND GUT RESECTION. *(E.)* Kinmonth, J. B.; Cox, S. J. (St. Thomas's Hosp. Med. Sch., London, England). *Br. J. Surg.* 61(8):589-593, 1974.

Three patients (a 14-yr-old girl and 2 men aged 52 and 54 yr) with primary lymphoedema of the small bowel and exudative enteropathy were investigated with mesenteric as well as pedal lymphography. Presenting symptoms included, resp., edema of legs and genitalia, edema of legs, and edema of genitalia. Two patients had fatty diarrhea. Gut protein loss, measured by the chromium chloride test, was 18.4, 10.8, and 2.5%, resp. Serum albumin measured 2.8, 2.9, and 3.8% while serum calcium was 4.9, 6.9, and 7.9%. Mesenteric lymphograms showed obstruction to gut drainage with no flow of dye to the cysterna chyli or thoracic duct. Additionally, lymphovenous shunting of dye to the liver was observed in 1 case. All 3 patients benefited from resection of the worst affected area of the gut (1-2 1/2 ft), followed in 2 cases by an end-to-end anastomosis. The protein loss in these patients was evidently due to exudation into the peritoneal cavity or into the gut lumen. The fatty diarrhea in 2 patients may have resulted from buildup of fat in lymph pathways, which became packed with chyle. At this stage fat absorption from the gut would slow or cease and fatty diarrhea ensue. The hypocalcemia may have arisen in association with the low plasma proteins. Additionally, excessive loss of calcium from the gut by a formation of insoluble soaps would be expected due to disordered fat absorption.

0451 RATE OF SYNTHESIS OF ALBUMIN IN RELATION TO SERUM LEVELS OF ESSENTIAL AMINO ACIDS IN PATIENTS WITH BACTERIAL OVERGROWTH IN THE SMALL BOWEL. *(E.)* Yap, S. H.; Hafkenscheid, J. C. M.; van Tongeren, J. H. M.; Trijbels, J. M. F. (Div. Gastroenterol., Univ. Nijmegen, Netherlands). *Eur. J. Clin. Invest.* 4(4):279-284, 1974.

In 11 patients bacterial overgrowth in the small intestine was confirmed by a breath test with 1-^{14}C-glycocholic acid and by counts of bacteria in jejunal juice. The patients had strictures of the jejunum or ileum due to Crohn's disease (5), small bowel diverticulosis (2), postgastrectomy diarrhea (2), celiac disease (1), and blind loop syndrome (1). Serum albumin concentrations were reduced (21.9-42.9 g/liter) in all but 1 patient. No relation could be demonstrated between the low albumin concentrations and the extent of gastrointestinal protein loss. The rate of albumin synthesis, determined by the ^{14}C-carbonate method, was significantly lower in the 11 patients compared with controls. The rate was positively correlated with serum levels of valine, isoleucine, leucine, and tryptophan: when the serum levels of these 4 essential amino acids were low, the rate of albumin synthesis was depressed. The reduction in serum amino acids was apparently the result of bacterial degradation of dietary amino acids, or malabsorption or low intake of amino acids.

0452 CARCINOID TUMORS OF THE GASTROINTESTINAL TRACT. *(E.)* Morgan, J. G.; Marks, C.; Hearn, D. (Louisiana State Univ. Sch. Med., New Orleans). *Ann. Surg.* 180(5):720-727, 1974.

0453 CARCINOID AND CARCINOID SYNDROME. *(Ger.)* Porst, H.; Naas, W.; Renger, F.; Dokert, B.; Platzbecker, H. (Carl Gustav Carus Med. Acad., Dresden, Germany). *Gesamte Inn. Med.* 29(15):635-638, 1974.

0454 LEIOMYOMA OF THE JEJUNUM AS THE CAUSE OF AN ACUTE ABDOMINAL CONDITION: REPORT OF A CASE. *(E.)* Goren, G.; Zelikowski, A.; Abu-Dalu, J.; Urga, I. (Beilinson Hosp., Petah Tiqua, Israel). *Dis. Colon Rectum* 17(6):782-785, 1974.

0455 PRIMARY ADENOCARCINOMA OF THE JEJUNUM AND COEXISTING PRIMARY ADENOCARCINOMA OF THE VERMIFORM APPENDIX: A CASE REPORT. *(E.)* Coblentz, M. G.; Filippone, D. R. (St. Barnabas Med. Cent., Livingston, N. J.). *Surgery* 77(1):160-164, 1975.

0456 MALIGNANT LYMPHOMA OF THE JEJUNUM IN A PATIENT WITH WISKOTT-ALDRICH SYNDROME. SURGICAL TREATMENT. *(E.)* Faraci, R. P., Hoffstrand, H. J.; Witebsky, F. G.; Blaese, R. M.; Beazley, R. M. (Natl. Cancer Inst., Bethesda, Md.). *Arch. Surg.* 110(2):218-220, 1975.

0457 NODULAR LYMPHOID HYPERPLASIA OF THE SMALL INTESTINE. *(E.)* Gerson, C. D.; Marshak, R. H.; Lindner, A. E. (Mt. Sinai Sch. Med., City Univ. New York, N.Y.). *Am. J. Gastroenterol.* 62(4): 371-376, 1974.

0458 AN EXPERIMENTAL MODEL FOR ASSESSING THE RADIOPERTECHNETATE DIAGNOSIS OF GASTRIC MUCOSA IN MECKEL'S DIVERTICULUM. *(E.)* Keramidas, D. C.; Coran, A. G.; Zaleska, W. (Los Angeles Cty.-Univ. South. California Med. Cent.). *J. Pediatr. Surg.* 9(6):879-884, 1974.

0459 THREE VARIETIES OF CONGENITAL DIVERTICULUM OF THE INTESTINE. *(E.)* Gray, S. W.; Akin, Jr., J. T.; Skandalakis, J. E. (Emory Univ. Sch. Med., Atlanta, Ga.). *Surg. Clin. North Am.* 54(6):1371-1377, 1974.

0460 SURGICAL PROBLEMS OF DUODENAL DIVERTICULA. *(Ger.)* Gober, I.; Strahberger, E. (2nd Surg. Clin., Univ. Vienna, Austria). *Wien. Klin. Wochenschr.* 86(15):433-436, 1974.

0461 PATHOPHYSIOLOGY AND CURRENT TREATMENT OF INTESTINAL OBSTRUCTION. *(E.)* Nadrowski, L. F. (no affil.). *Rev. Surg.* 31(6):381-407, 1974.

0462 SEQUELAE OF ILEUS TREATED SURGICALLY IN NEWBORNS OR INFANTS. *(Ger.)* Stroder, J.; Jeschke, R.; Fuchs, G.; Hardt, H.; Mingers, A. (Pediatr. Clin., Univ. Wurzburg, Germany). *Monatschr. Kinderheilk.* 122(7):707-709, 1974.

0463 SELECTIVE MASSIVE AMYLOIDOSIS OF SMALL INTESTINE. *(E.)* Griffel, B.; Man, B.; Kraus, L. (Meir Hosp., Kfar Saba, Israel). *Arch. Surg.* 110(2):215-217, 1975.

0464 THE ANATOMIC BASIS OF VASCULAR COMPRESSION OF THE DUODENUM. *(E.)* Akin, Jr., J. T.; Skandalakis, J. E.; Gray, S. W. (Piedmont Hosp., Atlanta, Ga.). *Surg. Clin. North Am.* 54(6):1361-1370, 1974.

0465 TRAUMATIC INJURIES TO THE DUODENUM: A REPORT OF 98 PATIENTS. *(E.)* Corley, R. D.; Norcross, W. J.; Shoemaker, W. C. (Hektoen Inst. Med. Res., Cook Cty. Hosp., Chicago, Ill.). *Ann. Surg.* 181(1):92-98, 1975.

0466 RETROPERITONEAL DUODENAL INJURY RESULTING FROM BLUNT ABDOMINAL TRAUMA. *(E.)* Doeven, J. J. (Surg . Clin., State Univ., Groningen, Netherlands). *Arch. Chir. Neerl.* 26(4):299-310, 1974.

0467 PNEUMOPERITONEUM SECONDARY TO ACUTE PERFORATION OF A TUBERCULOUS ULCER OF THE SMALL INTESTINE. A REPORT OF TWO CASES. *(E.)* Seliger, G.; Pinto, R. S. (New York Univ.-Bellevue Med. Cent., N. Y.). *Am. J. Gastroenterol.* 62(5):430-434, 1974.

0468 PRIMARY NONSPECIFIC SMALL BOWEL ULCERATION AS A SOURCE OF CHRONIC BLEEDING. REPORT OF A CASE AND REVIEW OF THE APPROACH TO LOCALIZATION OF THE SITE OF SMALL BOWEL HEMORRHAGE. *(E.)* Graham, D. Y.; Bynum, T. E. (Baylor Coll. Med., Houston, Tex.). *Am. J. Gastroenterol.* 62(4):350-355, 1974.

0469 BLEEDING FROM THE SMALL INTESTINE. *(Ger.)* Brunner, H. (Surg. Clin., Univ. Mainz, Germany). *Langenbecks Arch. Chir.* 337:569-575, 1974.

0470 TYPHOID ILEITIS WITH HEMORRHAGE. A CASE REPORT WITH NEW THERAPY. *(E.)* Collins, W. J.; Ball, R. E.; Dorsey, T. J. (St. Joseph Hosp., Orange, Calif.). *Am. J. Dig. Dis.* 20(1):94-96, 1975.

0471 INTESTINAL INFARCTION CAUSED BY ACUTE SPONTANEOUS THROMBOSIS OF THE PORTAL SYSTEM. *(Fr.)* Partensky, C.; Lesbros, F.; Saubier, E. C. (Edouard Herriot Hosp., Lyon, France). *Lyon Chir.* 70(4):261-264, 1974.

0472 ULTRASTRUCTURAL PATHOLOGICAL CHANGES IN INTESTINAL SUBMUCOSAL ARTERIOLES IN ANGIOTENSIN-INDUCED ACUTE HYPERTENSION IN RATS. *(E.)* Thorball, N.; Olsen, F. (Anat. Dep. C, Univ. Copenhagen, Denmark). *Acta Pathol. Microbiol. Scand.* [A] 82(6):703-713, 1974.

0473 STAGNANT LOOP SYNDROME RESULTING FROM SMALL-BOWEL IRRADIATION INJURY AND INTESTINAL BY-PASS. *(E.)* Swan, R. W. (William Beaumont Army Med. Cent., El Paso, Tex.). *Gynecol. Oncol.* 2(4):441-445, 1974.

0474 TREATMENT OF GROSS OBESITY BY JEJUNAL BYPASS. *(E.)* Gazet, J. C.; Pilkington, T. R. E.; Kalucy, R. S.; Crisp, A. H.; Day, S. (St. George's Hosp., London, England). *Br. Med. J.* 4(5940):311-314, 1974.

0475 SMALL BOWEL BYPASS IS APPLICABLE FOR ADOLESCENTS WITH MORBID OBESITY. *(E.)* White, J. J.; Cheek, D.; Haller, Jr., J. A. (Johns Hopkins Univ. Sch. Med., Baltimore, Md.). *Am. Surg.* 40(12):704-708, 1974.

0476 THERAPEUTIC ASPECTS OF THE PATHOLOGY OF INTESTINAL BACTERIA. *(Sp.)* Gaon, D. (no affil.). *Acta Gastroenterol. Latinoam.* 5:23-38, 1974.

See also, 0030, 0304, 0354, 0355, 0356, 0440, 0492, 0493, 0661, 0727, 0735, 0736, 0748, 0755, 0764.

SMALL INTESTINE

Malabsorption

0477 MORPHOLOGICAL AND BIOCHEMICAL CHANGES IN THE MUCOSA OF THE SMALL INTESTINE IN BLIND-LOOP SYNDROME. *(Ger.)* Bloch, R.; Menge, H.; Lorenz-Meyer, H.; Riecken, E. O. (Med. Clin., Univ. Marburg/Lahn, Germany). *Verh. Dtsch. Ges. Inn. Med.* 79:853-856, 1973.

Morphological and biochemical changes in the mucosa of a 3 cm long, self-filling blind-loop and in the distal and proximal adjoining segments of the rat small intestine were studied 10 to 12 days post operation. The blind-loop was inserted 15 cm below the ligament of Treitz. The measurement of the caprylate absorption revealed significant impairment of the transport function of the mucosa in the distal and proximal segments relative to a normal control. The ratio of the conjugated to free bile acids in the blind-loop and in the rest of the small intestine shifted form the normal value (11:1) to 1:1.7. A significant decrease occurred in the disaccharidase levels and in the enzyme activities. Morphometric examinations revealed pronounced zonal, hyperregenerative transformation of the mucosa, hypertrophic elongation of the villi and crypts, and an enlargement of the crypt zone at the expense of the villous zone. Hypertrophy of the absorptive cells was observed. Changes produced by bile in the mucosa reduce the cell life and lead to an immature absorptive epithelium.

0478 JEJUNAL BRUSH BORDER INJURY AND IMPAIRED SUGAR AND AMINO ACID UPTAKE IN THE BLIND LOOP SYNDROME. *(E.)* Giannella, R. A.; Rout, W. R.; Toskes, P. P. (Walter Reed Army Inst. Res., Washington, D.C.). *Gastroenterology* 67(5):965-974, 1974.

Small intestinal carbohydrate and amino acid uptake was studied in Walter Reed Carworth Farms male albino rats with blind loop syndrome to determine whether the intestinal epithelium was functionally or biochemically damaged. Rats were surgically equipped with either self-filling or self-emptying jejunal blind loops 10 cm distal to the ligament of Treitz. Absorption of ^{57}Co cyanocobalamin (5 ng) was measured 6-8 weeks after surgery. Normal rats and rats with self-emptying loops absorbed similar amounts of the dose, 64.2% and 62.8%, resp. Rats with self-filling loops absorbed only 25.5%. Both the blind loop and efferent jejunal segments of animals with self-filling blind loops contained significantly greater concentrations of aerobes and anaerobes. Maltase, sucrase, and lactase activities, and 3-*O*-methylglucose and L-leucine uptake were depressed in both the blind loop segment and jejunum distal to the loop in animals with self-filling blind loops. Both the site and severity of these abnormalities paralleled the degree of jejunal bacterial proliferation. After 6 days of either chloramphenicol or neomycin therapy, maltase and sucrase activities returned to normal, but 3-*O*-methylglucose and L-leucine uptake remained depressed. Chloramphenicol did not result in any change in concentration of total aerobes, coliforms, or total anaerobes in the jejunum. However, the obligate anaerobe bacteroides, which were present in very high concentrations, were eliminated. Neomycin therapy slightly reduced the concentration of total aerobes and eliminated aerobic streptococci and staphylococci. These findings indicate that the jejunal epithelial cell function may be abnormal in rats with the blind loop syndrome, resulting in impaired uptake of sugar and amino acid. It is likely that the jejunal brush border has been injured. The fact that this damage can be partly corrected by antibiotic therapy suggests a bacterial factor injurious to small intestinal mucosa.

0479 FAMILY STUDIES IN COELIAC DISEASE. *(E.)* Mylotte, M.; Egan-Mitchell, B.; Fottrell, P. F.; McNicholl, B.; McCarthy, C. F. (Reg. Hosp., Galway, Ireland). *Q. J. Med., New Series* 43(171): 359-369, 1974.

Peroral duodenojejunal biopsies were performed on 114 of 195 first degree relatives of 31 celiac patients (18 female, 13 male) in 28 families in Western Ireland. Only families in which the intestinal biopsy of the original case showed a flat or flat mosaic pattern when examined with the dissecting, and subtotal villous atrophy with the light microscope, and who showed a favorable clinical response to gluten withdrawal were accepted for the study. Jejunal or duodenal biopsies were done with a Crosby capsule or a modification of it. Mucosa fragments were examined by electron and dissecting microscopes and biochemical analysis. Enzymes analyzed were disaccharidases, leucine-proline hydrolase, enterokinase, and alkaline phosphatase. Quantitative determinations were made of serum immunoglobulins IgG, IgM and IgA by immunodiffusion. The enzyme levels and electron microscopic appearances of the histologically normal mucosa of celiac relatives was not different from that of a control group. Twelve of the 114 biopsies were characteristic of celiac disease. Birth order did not influence the incidence of celiac disease. The mode of inheritance is not typical of simple Mendelian inheritance but is compatible with a polygenic inheritance with a very high heritability or a dominant inheritance with a very low rate of expression in heterozygotes.

0480 LACK OF HARMFUL EFFECT OF OATS ON SMALL-INTESTINAL MUCOSA IN COELIAC DISEASE. *(E.)* Dissanayake, A. S.; Truelove, S. C.; Whitehead, R. (Radcliffe Infirm., Oxford, England). *Br. Med. J.* 4(5938):189-191, 1974.

Four celiac disease patients who had shown complete mucosal recovery after prolonged treatment of at least 1 yr with a strict gluten-free diet, volunteered to consume oats in addition to their gluten-free diet for 1 month. The oats were consumed at 40-60 g/day as porridge, oat cakes, or flap-jacks. The patients were studied by jejunal biopsy before and after the experimental period. All 4 remained symptom-free throughout the study as there was no appreciable change in the erythrocyte sedimentation rate or in the serum levels of iron, Vitamin B_{12} or folate. The serum IgA and IgM fell below normal in one patient after oats, but random fluctuations in serum immunoglobulins are not unusual in patients on a strict gluten-free diet. The appearances on dissecting and light microscopy remained unchanged. It is suggested that, from the limited knowledge of its chemical composition and antigenicity available currently, oat protein does not closely resemble wheat protein. If the gluten content of oats is related to the amide nitrogen, then 60 g of oats

would be equivalent to about 3-6 g of gluten. This study suggests that oat flour is not harmful to the jejunal mucosa of patients with celiac disease.

0481 BACTERIAL COLONISATION OF JEJUNAL MUCOSA IN ACUTE TROPICAL SPRUE. *(E.)* Tomkins, A. M.; Drasar, B. S.; James, W. P. T. (London Sch. Hyg. Trop. Med., England). *Lancet* 1(7898):59-62, 1975.

Numerous aerobic bacteria were closely associated with the mucosal layer of the proximal jejunum in 15 of 16 patients with acute tropical sprue. The patients (3 females and 13 males) registered an average weight loss of 11 kg. Enterobacteria were found in mucosa samples from 11 patients and streptococci were found in mucosa samples of 9. Viable bacteria were found in the luminal fluid of 9 patients: enterobacteria in 8 and streptococci in 3. Concentrations of bacteria in all cases were higher in mucosa samples than in luminal fluid. The species of enterobacteria present in 6 tropical sprue patients were *Alcaligines faecalis* (4), *Enterobacter aerogenes* (5), and *Hafnia* species (3). The incidence of streptococci in patients with acute tropical sprue were not related to mucosal abnormality. Bacteroides were not isolated from any patient. Two patients had lactobacilli in the luminal fluid; one had enterococci in the mucosa, but none had this in the luminal fluid. There were higher counts of enterobacteria in the mucosa of the 9 patients where the finger-shaped villi had been replaced by ridges and convolutions than in the 7 patients with milder changes. The presence of these enterobacteria was accompanied by an apparent decrease in the number of streptococci within the mucosa. The presence of enterobacteria was related to histological abnormality with high numbers of bacteria in the 7 patients with partial or subtotal villous atrophy. Tetracycline (250 mg/6 hr, p.o., for 4 weeks) either considerably reduced or erradicated the counts of enterobacteria within the mucosa. This reduction was accompanied by uniform improvement in mucosal appearance. One patient, although exhibiting initial improvement, failed to respond successfully to this therapy. Since clinical and biochemical improvement occurred on treatment with tetracycline when enterobacteria were eliminated from the mucosa, it is felt that these organisms may be responsible for persisting jejunal abnormalities in tropical sprue.

0482 SEASONAL OCCURRENCE OF OVERT AND SUBCLINICAL TROPICAL MALABSORPTION IN PUERTO RICO. *(E.)* Klipstein, F. A.; Corcino, J. J. (Trop. Malabsorpt. Unit, Univ. Rochester, N.Y.). *Am. J. Trop. Med. Hyg.* 23(6):189-196, 1974.

Among 96 rural residents of Puerto Rico who were evaluated in groups of 10-15/month from July through February, the prevalence of malabsorption of single and multiple substances and of severe jejunal morphologic abnormalities was significantly higher during the months of December through February than during the preceding 5 months. Of 27 persons hospitalized with overt tropical sprue during the course of the year, 14 (61%) also experienced the onset of their gastrointestinal symptoms during this 3-month winter period. Of 7 individuals who became symptomatic while living in the tropics, moved to New York City, and were subsequently hospitalized for tropical sprue, 6 (87%) noted the onset of symptoms during the period of December through March. In contrast, no such seasonal variation was evident among 16 expatriates from the West Indies who developed their initial symptoms of sprue while resident in New York City. Enterotoxigenic coliform bacteria, principally *Klebsiella pneumoniae* and *Enterobacter cloacae* were cultivated from the jejunum in 10 of 11 persons with tropical sprue who were investigated throughout the year. These organisms were also found in 2 of 3 rural residents examined in December through February, but in none of 6 other persons studied during the preceding 4 months. These observations point toward an environmental factor present in Puerto Rico during the period of December through February which is associated with a high prevalence of intestinal abnormalities in the general population, the onset of symptoms in persons with tropical sprue, and colonization of the small intestine by enterotoxigenic bacteria. The only environmental factor as yet identified was a change in dietary intake of fat commencing during early December in keeping with the month-long Christmas season in Puerto Rico. At this time the intake of pork and foodstuffs deep-fried in pork drippings increased markedly. The products are rich in unsaturated long chain fatty acids, principally oleic and linoleic acid. The presence of long-chain unsaturated fatty acids may contribute to changes in the bacterial population by altering the intestinal milieu to one unfavorable to the growth of bacteria normally resident there, with subsequent colonization by coliform bacteria.

0483 DRUG-INDUCED MALABSORPTION OF VITAMIN B_{12}. VI. MALABSORPTION OF VITAMIN B_{12} DURING TREATMENT WITH PHENFORMIN. *(E.)* Jounela, A. J.; Pirttiaho, H.; Palva, I. P. (Dep. Med., Univ. Onlu, Finland). *Acta Med. Scand.* 196(4):267-269, 1974.

The effect of long-term phenformin therapy on vitamin B_{12} absorption was studied in 25 diabetic patients (mean age 63.7 yr). The duration of diabetes in these patients was 7.1 yr and the length of drug therapy was 4.4 yr. Controls (11 additional patients) were treated with either chlorpropamide or tolbutamide. Diet was controlled in all patients. No patient was anemic. Of the 25 phenformin patients, 10 (40%) showed abnormally low excretion of radioactive vitamin B_{12} (4.4%) when studied without intrinsic factor. Control patients excreted 17.4%. Of the 10 phenformin patients with B_{12} malabsorption, 4 were unable to continue the study. Of the remaining 6, 5 again showed low excretion of labeled vitamin B_{12} when the test was repeated with intrinsic factor. Absorption of vitamin B_{12} was normalized in 4 of the 5 patients after discontinuation of phenformin treatment. In one of them this change was evident after an 8-day pause; in the others it required 2-8 weeks. The malabsorption noted in this study did not correlate with the

age of the patients, nor with the duration of their diabetes, nor with the duration of success of its treatment with phenformin.

0484 HISTOENZYMOLOGICAL STUDY OF THE INTESTINAL MUCOSA IN FIVE CASES OF WHIPPLE'S DISEASE. CHANGES DURING ANTIBIOTIC THERAPY. *(Fr.)* Ganter, P.; Marche, C.; Delesque, M.; Laumonier, R.; Metayer, J.; Cerf, M. (U.E.R. des Cordeliers, Paris, France). *Ann. Anat. Pathol.* 19(1):97-116, 1974.

Fragments of the intestinal mucosa were biopsied before treatment was begun in 2 cases and during treatment in 4 cases of Whipple's disease. Histological, ultrastructural, and histoenzymological studies, involving 21 hydrolases and oxidoreductases, were made for the enterocytes of the villi, the epithelium of the crypts, and the pathologic histiocytes of the lamina propria. The periodic acid-Schiff-positive macrophages of the intestinal epithelium showed abnormally high acid hydrolase activity (nonspecific acid phosphatase and β-D-glucuronidase). This finding is characteristic of Whipple's disease and subsides slowly with treatment. The activities of cytochrome oxidase and glucose-6-phosphatase in mucosal lamina propria macrophages were also elevated in 1 case. In 2 or 3 cases the phosphatase activity of the striated plateau was greatly diminished; decreases in the activities of nonspecific alkaline phosphomonoesterase, ATPase, 5'-nucleotidase, and thiamine pyrophosphatase may be implicated. It is concluded that the decrease in their activities accounts for the severe malabsorption. This abnormality disappears rapidly with treatment. Glucose-6-phosphatase, normally very active in the intestinal epithelium, was totally absent in 2 cases, and its activity varied in a 3rd case. This suggests disruption of the sodium pump, but the mechanism of the change is not known. This change was the slowest to respond to treatment. In one case, the activity of a number of other epithelial enzymes, particularly the dehydrogenases, was reduced.

0485 ENTEROKINASE ACTIVITY OF NORMAL AND FLAT DUODENAL MUCOSA. *(E.)* Rutgeerts, L.; Tytgat, G.; Mainguet, P.; Eggermont, E. (Dep. Pediatr., Univ. Louvain, Belgium). *Acta Gastroenterol. Belg.* 36(9):449-455, 1973.

The distribution of enterokinase along the small intestine of human adults with normal and flat intestinal mucosa was studied. A control group consisting of 10 male and 10 female subjects (17-51 yr old) had a morphologically normal intestinal mucosa and were free from gastrointestinal diseases. Eight male and 9 female patients (29-69 yr old) with malabsorption and a flat upper intestinal mucosa were studied before treatment; 4 male and 6 female patients (28-56 yr old) who had been treated with a gluten-free diet for at least 4 months were also studied. Intestinal biopsies were taken with a hydraulic biopsy tube. Two specimens, one for histological examination and one for enzyme assays, were obtained at 4 different levels of the duodenum. Duodenal juice was obtained in 14 control subjects and in 9 untreated patients. All enzyme studies were carried out immediately after homogenization or dilution to preserve enterokinase activity. Mucosal enterokinase activity was restricted almost exclusively to the duodenum and decreased from the proximal to the distal portion. Its distribution within the mucosa is confined to cells of the villous tips. In flat intestinal mucosa, enterokinase is less decreased than alkaline phosphatase or sucrase. Although most of the individual values fell within the normal range, the mean enterokinase activity in flat mucosa was significantly different from the mean value of normal mucosa. In patients with a flat intestinal mucosa, enterokinase activity of the duodenal juice was normal. Secondary enterokinase deficiency, therefore, seems not to be a prevalent problem in celiac disease.

0486 STUDY OF URINARY EXCRETION OF INDICAN IN PATIENTS SUBJECTED TO GASTRIC SURGERY. *(Sp.)* Rodrigo Gomez, J. M.; Aparisi Quereda, L.; Serra Desfilis, M. A.; Guix Garcia, J.; Tomas Ridocci, M.; Castellanos Escrig, G.; Parrilla Paricio, P.; Garcia-Conde Gomez, F. J. (Fac. Med., Valencia, Spain). *Rev. Esp. Enferm. Apar. Dig.* 42(3):311-322, 1974.

Urinary indican excretion was measured spectrophotometrically on 24-hr urine specimens from 78 patients, aged 25-65 yr, who had undergone the Billroth I procedure (22 cases), Billroth II procedure and vagotomy (35 cases), or truncal vagotomy and Heinicke-Mikulicz pyloroplasty (21 cases) 3 months to 5 yr before. Controls consisted of 37 normal subjects and patients with no evidence of gastrointestinal disease. All subjects received diets containing less than 80-90 g/day protein at this time. Fat determinations were performed on 24-hr stool specimens, taken on 3 consecutive days, from 36 of the patients who had undergone gastric surgery, and mean values for fecal fat excretion were calculated. A Schmidt-type diet was given for 5 days before fat determinations were made. Urinary indican excretion was significantly higher among patients who had undergone Billroth I and II procedures than among controls, while no difference was found between urinary indican excretion in controls and in patients who had been subjected to vagotomy and pyloroplasty. A significant correlation was found between urinary indican and fecal fat excretion. Bacterial overgrowth in the afferent loop might account for elevated urinary indican excretion in patients who had undergone the Billroth II procedure, while high indican values could be caused by intestinal bacterial proliferation secondary to impairment of pyloric function and decreased gastric secretion after the Billroth I procedure. In patients who had undergone vagotomy and pyloroplasty, steatorrhea is secondary to decreased pancreatic secretion and biliary dyskinesia caused by vagotomy, but this apparently does not produce bacterial overgrowth resulting in bile salt deconjugation and fat malabsorption as appears to occur in patients with Billroth II gastrectomies. Determinations of urinary indican excretion in patients who have undergone gastric surgery makes it possible to determine indirectly the extent of bacterial proliferation in the intestine which is secondary, in many cases, to blind-loop syndrome or

intestinal stenosis. The early diagnosis of these syndromes makes it possible to prevent the development of severe metabolic sequelae of gastric surgery.

0487 WHEN TO THINK OF CELIAC DISEASE. THE CLASSICAL FEATURES OF GLUTEN SENSITIVE ENTEROPATHY ARE OFTEN ABSENT. *(E.)* Brown, M. R.; Lillibridge, C. B. (Univ. Rochester Sch. Med., N.Y.). *Clin. Pediatr.* 14(1):76-82, 1975.

0488 ILEAL FUNCTION IN PATIENTS WITH COELIAC DISEASE. *(E.)* Silk, D. B. A.; Kumar, P. J.; Clark, M. L.; Dawson, A. M. (St. Bartholomew's Hosp., London, England). *Gut* 15(10):827-828, 1974.

0489 PRIMARY AMENORRHEA IN CELIAC DISEASE. TWO CASES. *(Fr.)* Netter, A. P.; Cahen, G. C. (Necker Hosp., Paris, France). *J. Obstet. Gynaecol. Biol. Repr.* 3(4):521-528, 1974.

0490 THE NATURE OF GLIADIN TOXICITY IN COELIAC DISEASE. *(E.)* Phelan, J. J. (Dep. Biochem., Univ. Coll., Galway, Ireland). *Biochem. Soc. Trans.* 2(6):1368-1370, 1974.

0491 COW'S MILK PROTEIN INTOLERANCE ASSOCIATED WITH A CHRONIC MALABSORPTION SYNDROME AND SEVERE INTESTINAL LESIONS SIMULATING CELIAC DISEASE. *(Fr.)* Deleze, G.; Nussle, D.; Megevand, A.; Gautier, E. (no affil.). *Helv. Paediatr. Acta* 33(suppl):24, 1974.

0492 VITAMIN B_{12} ABSORPTION IN INTESTINAL DISEASES. (COELIAC DISEASE, DERMATITIS HERPETIFORMIS, ULCERATIVE COLITIS, CROHN'S DISEASE, JEJUNOILEAL SHUNTING). *(E.)* Fausa, O. (Rikshosp., Oslo, Norway). *Scand. J. Gastroenterol.* [*Suppl.*] 9(29):75-79, 1974.

0493 MALABSORPTION OF VITAMIN B_{12} IN OBESE PATIENTS TREATED WITH JEJUNOILEAL SHUNT. *(E.)* Hippe, E.; Juhl, E.; Bruusgaard, A.; Korner, B.; Quaade, F.; Baden, H. (Bispebjerg Hosp., Copenhagen, Denmark). *Scand. J. Gastroenterol.* [*Suppl.*] 9(29):81-83, 1974.

0494 VITAMIN B_{12} ABSORPTION AFTER RESECTION OF ILEUM IN CHILDHOOD. *(E.)* Valman, H. B.; Roberts, P. D. (Queen Elizabeth Hosp. Child., London, England). *Arch. Dis. Child.* 49(12):932-935, 1974.

0495 THE ABSORPTION OF VITAMIN B_{12} IN THE BLIND-LOOP SYNDROME. *(E.)* Schjonsby, H. (Med. Dep. A, Univ. Bergen, Norway). *Scand. J. Gastroenterol.* [*Suppl.*] 9(29):65-70, 1974.

0496 DIAGNOSIS OF BLIND LOOP SYNDROME. *(Rus.)* Tamm, A. O.; Mikhlisaar, M. M. (Cent. Med. Res. Lab., Tartu, Univ., USSR). *Ter. Arkh.* 46:86-91, 1974.

0497 ABSORPTION OF SLOW-RELEASE IRON AND EFFECTS OF ASCORBIC ACID IN NORMAL SUBJECTS AND AFTER PARTIAL GASTRECTOMY. *(E.)* Baird, I. M.; Walters, R. L.; Sutton, D. R. (West Middlesex Hosp., England). *Br. Med. J.* 4(5943):505-508, 1974.

0498 WHIPPLE'S DISEASE: ETIOPATHOGENESIS, TREATMENT, DIAGNOSIS, AND CLINICAL COURSE. CASE REPORT AND REVIEW OF THE WORLD LITERATURE. *(E.)* Miksche, L. W.; Blumcke, S.; Fritsche, D.; Kuchemann, K.; Schuler, H. W.; Grozinger, K. H. (Community Hosp., Leverkusen, Germany). *Acta Hepatogastroenterol.* 21(4):307-326, 1974.

0499 WHIPPLE'S DISEASE MANIFESTED BY ARTHRITIS. *(Fr.)* Ghozlan, R.; Rampon, S.; Ryckewaert, A. (Lariboisiere Hosp., Paris, France). *Nouv. Presse Med.* 3(31):1935-1937, 1974.

0500 STUDIES ON TRYPTOPHAN CATABOLISM IN ACRODERMATITIS ENTEROPATHICA. *(E.)* Stachow, A.; Papuzinski, M.; Zalewski, T.; Skiendzielewska, A. (Warsaw Med. Sch., Poland). *Acta Derm. Venereol. (Stockh.)* 54(6):457-463, 1974.

0501 ACRODERMATITIS ENTEROPATHICA TREATED BY ZINC. *(E.)* Portnoy, B.; Molokhia, M. (Skin Hosp., Manchester, England). *Br. J. Dermatol.* 91(6):701-703, 1974.

See also, 0030, 0295, 0342, 0357, 0401, 0473, 0476, 0536, 0537, 0568, 0621, 0650, 0740, 0750.

LARGE INTESTINE

0502 COMBINATION OF CARCINOEMBRYONIC ANTIGEN AND GAMMA GLUTAMYL TRANSPEPTIDASE IN THE STUDY OF THE EVOLUTION OF COLORECTAL CANCER. *(E.)* Steele, L.; Cooper, E. H.; Mackay, A. M.; Losowsky, M. S.; Goligher, J. C. (Dep. Exp. Pathol., Cancer Res., Univ. Leeds, England). *Br. J. Cancer* 30(4):319-324, 1974.

Combinations of plasma carcinoembryonic antigen (CEA) values and serum γ-glutamyl transpeptidase activities were used to detect metastatic colorectal cancer and distinguish the sites of the metastases. Plasma CEA and serum γ-glutamyl transpeptidase levels were measured in 60 patients with primary tumors of the

colon or rectum and in 49 with metastatic colorectal cancer, in 17 of whom the primary tumor was also present. Similar measurements were made in 35 patients with histologically proven acute hepatitis, chronic hepatitis or cirrhosis. The normal value for CEA was 12.9 ng/ml. Normal glutamyl transpeptidase activities were 13.9 IU/liter. There was considerable overlap in CEA values between patients with colorectal cancer and those with non-malignant hepatic disease. CEA was plotted against the serum γ-glutamyl transpeptidase activities in patients presenting with primary colorectal cancers. A discriminant level of CEA of 100 ng/ml and a discriminant level of γ-glutamyl transpeptidase of 45 IU/liter were arbitrarily adopted. Of 43 tumors without evidence of spread outside the bowel and its local lymph nodes, or distant metastasis, 37 fell within the area bounded by these discriminants. Of the 17 tumors with hepatic metastases or local extension involving the peritoneum or pelvis, 14 had values on or outside the area. From 6 months to 8 yr after being clinically tumor free, 20 patients developed liver metastases. Using CEA values alone and a discriminant value of 100 ng/ml, only 3 of these 20 would not have been detected. Using the combination of parameters, all 20 would have been detected, and only 5 of 12 patients with local extension in the abdomen would have been false-positive for hepatic metastases. Using both these tests, clinically inapparent tumor in the liver can be detected before laparotomy or during follow-up. The serum γ-glutamyl transpeptidase activity indicates that the liver is diseased and the CEA value appears to discriminate between hepatomegaly due to metastatic cancer or benign hepatic disease.

0503 THE TREATMENT OF PEDUNCULATED ADENOMATOUS COLORECTAL POLYPS WITH FOCAL CANCER. *(E.)* Shatney, C. H.; Lober, P. H.; Gilbertsen, V. A.; Sonin, H. (Univ. Minnesota Health Sci. Cent., Minneapolis). *Surg. Gynecol. Obstet.* 139(6):845-850, 1974.

In 59 patients in whom the sole colorectal pathologic lesion was a pedunculated adenomatous polyp, 31 with *in situ* carcinoma were compared with 28 who had invasive carcinoma confined to the head of the tumor. In the 1st group (20 men 11 women, mean age 65 yr), 87% had the tumor in the rectosigmoid colon. Local excision was performed in 20 patients and extensive surgery in 11, with 1 death due to peritonitis following major surgery for removal of an *in situ* lesion. At the time of reporting 22 are alive and free of cancer of the colon. Of the 20 who underwent local surgery (2 by colonoscopic polypectomy) 15 remained alive and free of cancer. Of the 5 patients who died since surgery 4 lived for 5 or more years, and no evidence of cancer was found at the time of death. In the 2nd group, 17 patients (9 men 8 women, mean age 60 yr) had malignant changes limited to the distal portion of the head of the polyp and in 11 patients (8 men 3 women, mean age 64 yr) the carcinoma extended to the neck of the polyp. Diagnosis was established by barium enema examination in 15 cases and by proctoscopy in 13. No metastases were found in 14 patients who underwent laparotomy out of the group of 17. A metastasis in a pericolic lymph node was found in 1 of 9 patients who were operated for adenomatous polyps with carcinoma extending to the neck of the tumor. Of the group of 28 patients, 19 are alive and free of cancer of the colon (17 for at least 5 years post-treatment). Eight patients had only treatment of the lesions (colonic polypectomy in one case), and all are alive and free of disease, 5 of them for at least 5 years. It is concluded that the treatment of focally malignant pedunculated adenomatous polyps of the colon and rectum by colonoscopic removal is ideal when the malignancy does not extend further than the head of the tumor. Contraindications to local treatment would be the presence of lymphatic invasion, a highly undifferentiated cancer, and extremely short pedicle or extension of the malignancy to the neck of the polyp. Radical surgery is the treatment of choice for pedunculated adenoma with cancer extension to the base of the pedicle and for polypoid carcinoma.

0504 BLOOD MURAMIDASE ACTIVITY IN COLORECTAL CANCER. *(E.)* Cooper, E. H.; Turner, R.; Steele, L.; Goligher, J. C. (Dep. Exp. Pathol. Cancer Res., Univ. Leeds, England). *Brit. Med. J.* 3(5932):662-664, 1974.

Serum muramidase levels were measured in 166 tumor-free patients after resection of a colorectal cancer, 60 patients before resection of a localized primary adenocarcinoma of the colon or rectum, 39 patients with evidence of hepatic metastases, 29 patients with recurrence or local residual tumor spread in the pelvis, and 172 controls. Muramidase levels over 10 μg/ml were detected in 10-19% of the tumor-bearing patients, in 8.2% of the tumor free patients, and in only 1.7% of the controls (normal level 6.68 μg/ml). Repeated observations over periods of 3-12 weeks of 122 tumor-free patients and 26 patients with clinically detectable metastases or biochemical patterns suggesting metastases revealed 6.6% of the former group with muramidase levels over 10 μg/ml as compared to 38.3% with over 10 μg/ml muramidase for the latter group. There was no correlation between serum creatinine and muramidase levels in 35 patients chosen at random from those with raised muramidase levels. The long-term follow-ups indicated that raised muramidase levels may occur as a transient phenomenon in recurrent or metastatic disease. The reason for raised muramidase activity in some patients with cancer is not clear, though one possibility is that the increased activity is a reflection of a host-tumor reaction, perhaps a stimulation of the reticuloendothelial system.

0505 ACTION OF DIFFERENT BRAN PREPARATIONS ON COLONIC FUNCTION. *(E.)* Kirwan, W. O.; Smith, A. N.; McConnell, A. A.; Mitchell, W. D.; Eastwood, M. A. (Dep. Clin. Surg., Univ. Edinburgh, Scotland). *Brit. Med. J.* 4(5938):187-189, 1974.

The effectiveness of fine and coarse bran preparations in lowering intraluminal pressure and decreasing transit time in patients with constipation and diverticular disease was investigated. Fine bran

(10 g twice daily for 4 weeks) was given to 5 patients, 3 with radiologically proven diverticular disease, and 2 with constipation. Coarse bran was given in the same dosage to 9 patients with diverticular disease. Basal, post-food, and post-neostigmine motility indices for the coarse-bran patients decreased from 892.8 to 648.7, 1513.2 to 446.1, and 2120.0 to 1216.8, resp. Gastrointestinal transit time was lowered from 93.4 to 57.9 hr. Basal, post-food, and post-neostigmine motility indices for the fine-bran patients increased from 1181 to 1269, 1429 to 2534, and 4075 to 4661, resp. Gastrointestinal transit time fell from 98 to 86 hr. In 4 patients first given fine bran and then changed to coarse bran, the mean transit time after 4 weeks of coarse bran was lowered from 96 hr to 51 hr. Basal, post-food, and post-neostigmine motility indices in this group changed froom 1202 to 303, 2369 to 1459, and 4726 to 2746. The greatest effect of coarse bran in reducing intraluminal pressure and decreasing transit time appears to be due to its greater water-holding capacity which is a function of particle size, thus making it preferable for the treatment of colonic disorders.

0506 EXPERIENCE WITH A CHOLINESTERASE HISTOCHEMICAL TECHNIQUE FOR RECTAL SUCTION BIOPSIES IN THE DIAGNOSIS OF HIRSCHSPRUNG'S DISEASE. *(E.)* Trigg, P. H.; Belin, R.; Haberkorn, S.; Long, W. J.; Nixon, H. H.; Plaschkes, J.; Spitz, L.; Willital, G. H. (Hosp. Sick Child., London, England). *J. Clin. Pathol.* 27(3):207-213, 1974.

A cholinesterase histochemical technique was applied to rectal suction biopsies in an attempt to make the submucosal nervous tissue more conspicuous and also to evaluate the significance of mucosal cholinesterase activity in the diagnosis of Hirschsprung's disease. A total of 160 biopsies were taken from 101 patients (aged 2 days to 17 yr) and processed for cholinesterases. All patients had presented with a clinical picture suggestive of Hirschsprung's disease which was ultimately confirmed by definitive surgery in 33 and excluded in the remainder. The biopsy was immediately spread submucosa-down onto a small block of animal spleen or liver and quick frozen in hexane. The sample was cut at 15 μ in a plane perpendicular to the flattened submucosal surface. Slides were air-dried for a few min, immersed for 30 sec in 10% neutral formalin at room temperature and washed in distilled water. They were incubated for 3 hr at 37 C using a medium described by Karnovsky and Roots, and an acetate buffer at pH 6.0 and s-acetylthiocholine iodide as substrate. Inhibitors were not routinely used, so the histochemical reaction presumably demonstrated both true (acetyl-) and pseudocholinesterase activity. After rinsing in distilled water, slides were counterstained in Ehrlich's hematoxylin to show the nuclei, differentiated in 1% acid alcohol, blued in running tap water, dehydrated, cleared in xylol and mounted in Canada balsam. The end product of the histochemical reaction is a finely granular and highly refractile deposit of red-brown copper ferrocyanide. Localization was good. The method proved at least as reliable as experienced assessment of paraffin hematoxylin-eosin sections, and appeared to offer the advantages of reduced scanning fatigue, and superior demonstration of the increased cholinesterase-positive nerves in Hirschsprung's disease. It was not possible to base a diagnosis on mucosal cholinesterase activity.

0507 MITOTIC ACTIVITY OF EPITHELIAL CELLS IN IN WOUNDED RECTAL MUCOUS MEMBRANE. *(E.)* Reeve, D. R. E. (R. Coll. Surg. England, London). *J. Anat.* 118(2):231-239, 1974.

Patterns of mitotic activity in the epithelial cells of the rectal mucous membrane at a wound margin were studied using female Wistar rats. Ulcers were made by excising small portions of the rectal mucous membrane. Within 24 hr the mitotic number had increased 2-fold over controls and peaked at day 2, with a mitotic number 5 times controls. The level fell rapidly the next day, but rose to a smaller peak of activity on day 4. During the following 3 days the levels fell to pre-peak values and remained constant. A third large peak appeared on the 18th or 19th day, falling by day 20. A 4th, smaller peak occurred on day 24 which also dropped a day later. Autoradiography in a further series of animals confirmed that there is after wounding an obvious increase in mitotic activity which does not occur as a simple rise and fall but as a series of peaks occurring at reasonably constant times after wounding (2, 11-12, 18-19, and 22-24 days). There was also an overall rise in the level of mitotic activity up to day 14, after which the level fell to almost normal values. This peak and trough pattern suggests that one effect of injury is to induce a temporary synchrony in the cell cycle so that cell division occurs *en masse*. Although mitotic cells are usually confined to the basal parts of the colonic glands, as the mitotic number approached a peak, mitoses were found in the middle parts of the glands as well.

0508 PERFORATIVE CARCINOMA OF COLON AND RECTUM. *(E.)* Welch, J. P.; Donaldson, G. A. (Massachusetts Gen. Hosp., Boston). *Ann. Surg.* 180(5):734-740, 1974.

0509 CARCINOMA OF THE RECTUM. *(Gr.)* Xanthakis, D. S. (no affil.). *Iatriki* 24(1):19-30, 1973.

0510 CARCINOMA OF THE COLON, RECTUM, AND ANAL CANAL IN YOUNG PATIENTS. *(E.)* Recalde, M.; Holyoke, E. D.; Elias, E. G. (Roswell Park Mem. Inst., Buffalo, N.Y.). *Surg. Gynecol. Obstet.* 139(6):909-913, 1974.

0511 100% OXYGEN INHALATION AND 5-FLUOROURACIL (NSC-19893) THERAPY FOR LARGE BOWEL CARCINOMA. *(E.)* Moertel, C. G.; Schutt, A. J.; Reitemeier, R. J.; Hahn, R. G. (Mayo Clin., Rochester, Minn.). *Cancer Chemother. Rep.* 58(6):951-952, 1974.

0512 STEROID NUCLEAR DEHYDROGENATION AND COLON CANCER. *(E.)* Hill, M. J. (Cent. Public Health Lab., London, England). *Am. J. Clin. Nutr.* 27(12):1475-1480, 1974.

0513 ADENOACANTHOMA OF THE APPENDIX. *(E.)* Schulte, W. J.; Pintar, K.; Schmahl, T. (Wood VA Cent., Milwaukee, Wis.). *J. Surg. Oncol.* 6(2):93-96, 1974.

0514 ADENOCARCINOMA OF THE VERMIFORM APPENDIX. *(E.)* Forsgren, L.; Molin, K.; Rieger, A. (Danderyd Hosp., Sweden). *Acta Chir. Scand.* 140(6): 486-490, 1974.

0515 CANCER RISK FOLLOWING APPENDECTOMY. *(Ger.)* Grosse, H. (Am Sund Reg. Hosp., Stralsund, Germany). *Arch. Geschwulstforsch.* 43(4):358-363, 1974.

0516 COLONIC ISCHEMIA AFTER PROSTHETIC REPLACEMENT OF THE ABDOMINAL AORTA AND OTHER FORMS OF ISCHEMIC COLITIS. *(Ger.)* Rausis, C.; Mirkovitch, V.; Robinson, J. W. L.; Roenspiess, U.; Saegesser, F. (Surg. Clin. A, Univ. Lausanne, Switzerland). *Praxis* 63(18):545-549, 1974.

0517 ISCHEMIC COLITIS: REVISION OF ITS ANATOMICAL AND CLINICAL CLASSIFICATION WITH REGARD TO ONE CASE OF COLONIC VASCULITIS. *(Sp.)* Lain Martinez, P.; de Villalobos, E.; Nadal Capara, J.; Ramirez Guedes, Guedes, J.; Oliva, H. (Jimenez Dias Found., Madrid, Spain). *Rev. Clin. Esp.* 129(6):561-566, 1973.

0518 LAXATIVE ABUSE. *(E.)* Cummings, J. H. (Cent. Middlesex Hosp., London, England). *Gut* 15(9):758-766, 1974.

0519 PNEUMATOSIS CYSTOIDES INTESTINALIS. A CASE DIAGNOSED BY MEANS OF RECTOSCOPY, WITH BIOPSY AND A SUMMARY OF THE DANISH OBSERVATIONS. *(E.)* Kristensen, J. K. (Cty. Hosp., Braedstrup, Denmark). *Acta Chir. Scand.* 140(6):501-504, 1974.

0520 THE HIGH FIBRE DIET IN THE TREATMENT OF DIVERTICULAR DISEASE OF THE COLON. *(E.)* Painter, N. S. (Manor House Hosp., London, England). *Postgrad. Med. J.* 50(588):629-635, 1974.

0521 A TRANSINTESTINAL GUIDE FOR FIBEROPTIC COLONOSCOPY IN DIFFICULT CASES. *(E.)* Rogers, B. H. G. (Henrotin Hosp., Chicago, Ill.). *Gastrointest. Endosc.* 21(2):62-65, 1974.

0522 SIGNIFICANCE OF A NEW THERAPEUTIC DRUG, DIOSMINE, IN PROCTOLOGY. *(Fr.)* Peeters, J. P. (Brugmann Univ. Hosp., Brussels, Belgium). *Brux. Med.* 53(10):591-594, 1973.

See also, 0291, 0308, 0334, 0342, 0358, 0359, 0360, 0452, 0455, 0476, 0546, 0548, 0736, 0754, 0755, 0790, 0791, 0793.

LARGE INTESTINE

Ulcerative Colitis

0523 NECROTIZING VASCULITIS AND ULCERATIVE COLITIS. *(E.)* Wackers, F. J. T.; Tytgat, G. N.; Vreeken, J. (Dep. Intern. Med., Univ. Amsterdam, Netherlands). *Br. Med. J.* 4(5936):83-84, 1974.

A 26-yr-old man was treated with coumarin for 6 months because of migrating thrombophlebitis. On the second day after stopping the coumarin, the patient became feverish and developed painful bluish spots on his thighs; some of these disappeared spontaneously, but two days later a more extensive bluish area appeared in the inner aspect of the left thigh and this last lesion developed a central hemorrhagic necrosis of the skin. Coumarin was again prescribed, but was stopped after 3 days because of hematuria and bloody stools. Laboratory tests showed the erythrocyte sedimentation rate was 73 mm in the first hour, hemoglobin was 9 g/100 ml and WBC 20,000/mm^3. Fibrin degradation products were increased and Thrombotest time (Owren) was prolonged. All other coagulation values were normal. Benzidine test for occult blood in the feces was strongly positive. Arthritic, polyarthritic and purpuric lesions apparently vasculitic in origin developed in various locations and it was felt that the patient was suffering from a systemic or underlying disease. Careful questioning disclosed several episodes of diarrhea in previous years with blood in the stools during the last 2 months of coumarin treatment. A sigmoidoscopy was performed. The bowel mucosa was edematous and swollen, covered by thick mucopurulent exudate and showing numerous hemorrhagic spots and tiny superficial erosions, the picture strongly suggestive of severe ulcerative colitis. Biopsy specimens confirmed this diagnosis. The patient was initially treated with 3 g sulphasalazine, 50 mg prednisone and a short course of ampicillin. I.v. heparin was administered for 10 days. The areas of skin necrosis were debrided and healed by granulation or with the help of skin grafts. A sigmoidoscopy performed 1 1/2 months after treatment revealed an almost normal mucosa. The prednisone was reduced to 15 mg/day. On follow-up 9 months later the patient was found in excellent condition clinically and by sigmoidoscopy.

0524 ENDOSCOPIC RETROGRADE CHOLANGIOPANCREATOGRAPHY IN THE DIAGNOSIS OF JAUNDICE ASSOCIATED WITH ULCERATIVE COLITIS. *(E.)* Elias, E.; Summerfield, J. A.; Dick, R.; Sherlock, S. (R. Free Hosp., London, England). *Gastroenterology* 67(5): 907-911, 1974.

Endoscopic retrograde cholangiopancreatography was performed in 4 jaundiced ulcerative colitis patients. In a 30-yr-old male, a liver biopsy 4 yr earlier had been thought to show primary biliary cirrhosis and a recent attempt at percutaneous transhepatic cholangiography had failed. Retrograde cholangiopancreatography revealed a short extremely irregular stricture high in the common bile duct. The normal caliber and outline of the duct below the stricture favored diagnosis of cholangiocarcinoma, rather than sclerosing cholangitis. The shortness of the segment and the severity of obstruction indicated immediate surgery and carcinoma was successfully resected. In a 35-yr-old woman and a 21-yr-old man, endoscopic retrograde cholangiopancreatography revealed changes typical of sclerosing cholangitis, including multiple strictures with beading of ducts between the narrowed segments, and involvement of both the intrahepatic and extrahepatic systems. In both these cases surgery was averted. In a 16-yr-old jaundiced male, the depth of icterus made any attempt at oral or i.v. cholangiography impossible, and a clotting disorder precluded either liver biopsy or percutaneous cholangiography. A retrograde cholangiogram revealed a normal biliary tree, excluding extrahepatic obstruction. Liver biopsy just prior to discharge showed changes of a long-standing, probably viral, hepatitis. Endoscopic retrograde cholangiopancreatography is of particular value in jaundiced patients with ulcerative colitis, not only in demonstrating extrahepatic obstruction when present, but also in showing whether surgical intervention is likely to benefit the patient. Thus patients with features of cholestasis may be spared liver biopsy, unsuccessful percutaneous transhepatic cholangiography, and hazardous biliary surgery.

0525 HYPOSPLENISM AND ULCERATIVE COLITIS. *(E.)* Ardeman, S.; Bevan, G. (Edgware Gen. Hosp., England). *Lancet* 2(7880):588, 1974.

PANCREAS

0526 NONFUNCTIONING NONCHROMAFFIN PARAGANGLIOMA OF THE PANCREAS. *(E.)* Cope, C.; Greenberg, S. H.; Vidal, J. J.; Cohen, E. A. (Albert Einstein Med. Cent., Philadelphia, Pa.). *Arch. Surg.* 109(3): 440-442, 1974.

Barium study of the gastrointestinal tract of a 72-yr-old woman revealed a pancreatic mass that caused elevation and widening of the duodenal loop. The same symptomless tumor had been noted 2 yr earlier. Angiography showed a well-rounded paraganglioma (11 x 14 cm), the blood supply of which derived equally from the pancreaticoduodenal and omental arteries and branches of the right colic artery from the superior mesenteric artery. The tumor and a small portion of adjacent pancreas were removed. Microscopical examination indicated a very cellular, well-encapsulated neoplasm. In the area traversing the transition from pancreas to neoplasm, there was some invasion of the capsule as well as invasion of large vascular sinuses by neoplasm. The tumor did not stain with chrome salts or glutaraldehyde and was diagnosed as a juxtapancreatic nonchromaffin paraganglioma. This is only the 4th reported case of such a tumor associated with the pancreas. The patient's chance of cure is excellent due to her age and the lack of satellite tumors.

0527 PANCREATIC DISEASE. *(E.)* Kafka, E. C.; Kalser, M. H. (Univ. Miami Sch. Med., Fla.). *Postgrad. Med.* 57(1):140-152, 1975.

0528 THE DIAGNOSIS OF PANCREATIC CARCINOMA. *(Ger.)* Loffler, A.; Stadelmann, O.; Miederer, S. E.; Sobbe, A. (Med. Polyclin., Univ. Bonn, Germany). *Dtsch. Med. Wochenschr.* 99(40):1976-1983, 1974.

0529 REPLACEMENT OF PORTAL VEIN DURING PANCREATECTOMY FOR CARCINOMA. *(E.)* Norton, L.; Eiseman, B. (Denver Gen. Hosp., Colo.). *Surgery* 77(2): 280-284, 1975.

0530 PANCREATICO-DUODENAL RESECTION AND BY-PASS OPERATION IN PATIENTS WITH CARCINOMA OF THE HEAD OF THE PANCREAS, AMPULLA, AND DISTAL END OF THE COMMON DUCT. *(E.)* Hertzberg, J. (Ulleval Hosp., Oslo, Norway). *Acta Chir. Scand.* 140(7):523-527, 1974.

0531 SPONTANEOUS RUPTURE OF A PANCREATIC PSEUDOCYST INTO THE COLON. *(E.)* Barnardo, D. E.; Grogono, J. L.; Parks, A. G. (London Hosp., England). *Am. J. Dig. Dis.* 19(12):1165-1167, 1974.

0532 PANCREATIC FISTULAS. *(Ger.)* Mangold, G. (Surg. Clin., Univ. Mainz, Germany). *Langenbecks Arch. Chir.* 337:127-130, 1974.

0533 PANCREAS INJURIES IN BLUNT ABDOMINAL TRAUMA. EXPERIENCE WITH DIAGNOSIS AND TREATMENT IN 20 PATIENTS. *(E.)* Cardozo, M. L. (Surg. Clin., State Univ., Groningen, Netherlands). *Arch. Chir. Neerl.* 26(4):289-297, 1974.

0534 DISORDERS IN PANCREATIC FUNCTION OF TOXIC CHEMICAL ORIGIN. *(Rus.)* Kleiner, A. I.; Vasilenko, N. M.; Sonkin, I. S.; Ostrovskaia, I. S.; Iashina, L. N.; Liss, N. L. (Inst. Ind. Hyg. Occup. Dis., Kharkov, USSR). *Gig. Tr. Prof. Zabol.* (10):15-18, 1974.

0535 INSULAR HYPERPLASIA AND HYPOGLYCAEMIA: A REVIEW OF THE PROBLEM. *(E.)* Carboni, M.; Patrassi, N. (Surg. Clin. II, Univ. Rome, Italy). *Rend. Gastroenterol.* 5(3):235-236, 1973.

0536 ABSORPTION OF VITAMIN B_{12} IN PANCREATIC INSUFFICIENCY. *(E.)* von der Lippe, G. (Med. Dep. A, Univ. Bergen, Norway). *Scand. J. Gastroenterol.* [*Suppl.*] 9(29):71-73, 1974.

0537 VITAMIN B_{12} MALABSORPTION IN EXOCRINE PANCREATIC INSUFFICIENCY. *(Ger.)* Aenishanslin, H. W.; Stalder, G. A. (Med. Polyclin., Univ. Basel, Switzerland). *Dtsch. Med. Wochenschr.* 99(41):2033-2035, 1974.

See also, 0098, 0116, 0335, 0336, 0337, 0361, 0362, 0363, 0364, 0365, 0366, 0422, 0648, 0655, 0673, 0696, 0702.

PANCREAS

Pancreatitis

0538 ACUTE PANCREATITIS IN PREGNANCY. *(E.)* Jouppila, P.; Mokka, R.; Larmi, T. K. I. (Oulu Univ. Cent. Hosp., Finland). *Surg. Gynecol. Obstet.* 139(6):879-882, 1974.

Over a 2 yr period 8 pregnant patients were admitted with acute pancreatitis. This represented an incidence of 1 for every 2,302 deliveries. Acute pancreatitis was contracted between 11-29 weeks of pregnancy. The mothers were 23-35 yr old and para 1, 2, 3 and 4. On admission 3 of the patients were thought to have an acute condition of the abdomen. Jaundice was present in 6 patients. Laboratory tests showed elevated blood and urine amylase levels in all patients; serum lipase levels were elevated in 4 patients and serum calcium levels were subnormal in 4. Mild leukocytosis was present in 4 and moderate leukocytosis in the others. In 6 patients, the levels of serum bilirubin or serum alkaline phosphatase or both were elevated, and in 4 the SGOT levels were high. After diagnosis was made, the patients were started on nasogastric suction and intravenous fluid therapy. Antibiotics (ampicillin in 7 and penicillin G with streptomycin in 1), an antispasmodic (Baralgin), and anticholinergic drugs (either atropine s.c. or propantheline bromide i.m.) were used in the treatment. Three patients had recurrence of pancreatitis during the same pregnancy and 1 was operated for obstructive jaundice. Cholelithiasis was present in 5 of the patients. All 8 patients survived, but 3 fetuses were lost. The role of pregnancy in acute pancreatitis is unclear, but it has been noted that the gallbladder becomes larger and empties less effectively than usual, possibly due to changes in flow conditions of the bile caused by high progesterone levels. Cholelithiasis is still considered the most common etiological factor and surgical intervention is indicated even during pregnancy when unremitting pancreatitis is dangerous to life.

0539 SURGICAL MANAGEMENT OF ACUTE AND CHRONIC PANCREATITIS. *(E.)* Hermann, R. E. (Cleveland Clin., Ohio). *Clin. Med.* 81(8):24-26, 1974.

0540 PANCREATITIS. A PROSPECTIVE CLINICAL EVALUATION OF 100 CASES AND REVIEW OF THE LITERATURE. *(E.)* Olsen, H. (Providence Hosp., Oakland, Calif.). *Am. J. Dig. Dis.* 19(12):1077-1090, 1974.

0541 HEMORRHAGE IN PANCREATIC INFLAMMATORY PSEUDOCYST. (REVIEW OF LITERATURE AND REPORT OF A CASE OF MASSIVE MELENA CAUSED BY HEMORRHAGE INTO A PANCREATIC PSEUDOCYST COMMUNICATING WITH THE PANCREATIC DUCTS). *(It.)* Pasquali, E.; Corsi, A. (Prov. Gen. Hosp., Conegliano, Italy). *Chir. Gastroenterol.* 7(4):554-562, 1973.

0542 PATHOGENESIS OF ACUTE PANCREATITIS. *(Gr.)* Polyzos, J. (Dep. Pathol., Natl. Capodistrian Univ., Athens, Greece). *Iatriki* 23(6):453-469, 1973.

0543 RECENT ADVANCES IN THE TREATMENT OF ACUTE HEMORRHAGIC NECROTIZING PANCREATITIS. *(Fr.)* Hollender, L. F. (Univ. Hosp. Cent., Strasbourg, France). *Bull. Acad. Natl. Med. (Paris)* 158(2):130-138, 1974.

0544 ACUTE NECROTIZING PANCREATITIS. AN EXPERIMENTAL MODEL. *(E.)* Bawnik, J. B.; Orda, R.; Wizniter, T. (Ichilov Hosp., Tel Aviv, Israel). *Am. J. Dig. Dis.* 19(12):1143-1147, 1974.

0545 ON 135 CONSECUTIVE CASES OF ACUTE PANCREATITIS. *(Fr.)* Simard, E.; Gagnon, G.; Beaudry, E.; Brisson, F.; Bergeron, C.; Charbonneau, A. (Hosp., Chicoutimi, Canada). *Union Med. Can.* 103(9):1578-1581, 1974.

0546 INTESTINAL FISTULA COMPLICATING PANCREATIC ABSCESS. *(E.)* Mason, H. D. W.; Forgash, A.; Balch, H. H. (District Columbia Gen. Hosp., Washington). *Surg. Gynecol. Obstet.* 140(1):39-43, 1975.

0547 COMPLICATIONS OF CHRONIC PANCREATITIS. *(E.)* Lewis, T. D. (Repatriation Gen. Hosp., Heidelberg, Australia). *Aust. NZ J. Med.* 4(5):518-521, 1973.

0548 EXTRINSIC STENOSIS OF THE LEFT COLONIC ANGLE BY CHRONIC PANCREATITIS. *(Fr.)* Briet, S.; Favre, J. P.; Sgro, J. C.; Viard, H. (Bocage Hosp., Dijon, France). *Lyon Chir.* 70(5):323-324, 1974.

0549 AMYLASE PLEURISY AND SPONTANEOUS RUPTURE OF THE SPLEEN IN CHRONIC CALCIFYING PANCREATITIS. *(Fr.)* Turiaf, J.; Battesti, J. P.; Pessayre, J. R.; Boutelier, P.; de Tayrac, R. (Bichat Hosp., Paris, France). *Ann. Med. Interne (Paris)* 125(6/7): 527-532, 1974.

See also, 0098, 0333, 0337, 0338, 0371, 0527.

LIVER AND BILIARY TRACT

0550 ESTIMATION OF THE FUNCTIONAL RESERVE OF HUMAN LIVER. *(E.)* Moody, F. G.; Rikkers, L. F.; Aldrete, J. S. (Univ. Utah Med. Cent., Salt Lake City.). *Ann. Surg.* 180(4):592-598, 1974.

Functional hepatic reserve was determined in 32 patients with known liver or biliary tract disease (8 with alcoholic cirrhosis, 5 with biliary or postnecrotic cirrhosis, 5 with portal decompression, 14 with a variety of liver disturbances) employing kinetic analysis of hepatic removal of indocyanine green. The initial removal rates of incremental i.v. doses of indocyanine green (0.5, 1.0, and 5.0 mg/kg) were plotted as a reciprocal against the inverse of dose (Lineweaver-Burk plot) to provide a means of determining maximal removal rate from submaximal doses. Blood was withdrawn before and at 5, 10, and 15 min intervals following administration of the dye. The maximal removal rate, which was 3.40 mg/kg/min in 10 control patients, was only 0.24 mg/kg/min in 8 patients (5 male and 3 female, ages 35-58 yr) with alcoholic cirrhosis. Portasystemic shunting did not further influence the maximum removal rate of the dye, and infiltrative liver disease had only a mild depressive effect on this function. The results demonstrate that hepatic function can be precisely quantitated by classical enzyme kinetics (Michaelis-Menten). If the maximum removal rate of indocyanine green from the blood (*via* the liver) is an estimate of protein receptor mass for organic anions, then the technique may allow an indirect means for quantitating hepatocytes even in the presence of changes in blood flow or hepatic function. The profound depression in this removal rate observed in patients with alcoholic cirrhosis is consistent with the progressive loss in hepatic mass associated with this disease.

0551 A PRACTICAL APPROACH TO QUANTITATE HEPATIC EXCRETORY FUNCTION. *(E.)* Bircher, J.; Hacki, W. (Dep. Clin. Pharmacol., Univ. Bern, Switzerland). *Yale J. Biol. Med.* 3(47):196-201, 1974.

In a group of patients with various liver diseases and in normal control subjects, the 3 BSP tests used to assess hepatic function, namely BSP retention, BSP plasma disappearance curve, and BSP transport maximum (BSP T_m), were carried out in one session. The BSP disappearance curves were measured within 30 sec. after the i.v. injection of 5 mg/kg BSP. Blood samples were obtained at 2-5 min intervals up to 45 min beginning 3 min after injection. The last sample was used to calculate BSP retention assuming an initial concentration of 10 mg/100 ml. The BSP T_m was determined indirectly with the two-infusion method. The logarithm of the BSP retention correlated linearly with the excretory capacity of the liver as revealed by the BSP T_m. There was a close correlation between the logarithms of the second component (k_2) of the disappearance curve and the BSP T_m, suggesting that k_2 is mainly determined by the excretory capacity of the liver. It is suggested that the use of BSP k_2 and of BSP retention U should be valuable in the follow-up of patients treated for chronic liver disease. Because of its better established physiological basis, the BSP k_2 is directly applicable to the individual patient and, therefore, may be preferred.

0552 STUDY OF LIVER FUNCTION IN CHILDREN USING ^{131}I-LABELED BENGAL ROSE. *(Rus.)* Ermakova, T. M. (I.M. Sechenov 1st Moscow Med. Inst., USSR). *Pediatriia* (12):19-20, 1973.

Studies of 22 patients with systemic lupus erythematosus by ^{131}I-labeled bengal rose showed serious hepatic absorption and secretion disorders. Half-life periods varied between 6-102 min for absorption (normally 6-12 min); 19-1225 min for excretion (80-120); 4-570 min for purification (6-20): vascularization ranged between 40 and 98% (98-99%, normally). Findings indicate a serious subclinical liver deficiency involving the vascular system. Standard biochemical tests did not reveal any significant changes of the liver function.

0553 DETERMINATION OF THE SERUM GUANASE ACTIVITY IN DISEASES OF THE HEPATIC PARENCHYMA.

(Ger.) Kramp, B.; Teichmann, W.; Rehpenning, W. (Dep. Gastroenterol., Univ. Rostock, Germany). *Dtsch. Z. Verdau. Stoffwechselkr.* 33(1):11-16, 1973.

The serum guanase activity was measured by Kramp's modified method in 21 patients with acute viral hepatitis, 16 with chronic aggressive hepatitis, and in 21 with cirrhosis to determine the diagnostic value of this parameter. Increased serum guanase activity was measured in 18 cases of acute viral hepatitis during the first 10 days of the disease, while the SGOT and SGPT activities showed increase in 17 cases each. Pathologically increased guanase activity, and increased SGOT and SGPT activities were found in 13 and 7 cases of chronic aggressive hepatitis, resp. Pathological increase of the guanase activity was observed in 15 cases of cirrhosis, while the SGOT and SGPT activities were pathologically high in 12 and 7 cases, resp. Serum guanase activities measured in acute viral and chronic aggressive hepatitis, and cirrhosis were in the ranges of 0-155.3 mU/ml (average 35.8 mU/ml); 0-49.8 mU/ml (average 13.8 mU/ml), and 0-2.9 mU/ml, resp. The findings indicate that the serum guanase activity test fails to offer special advantages over the transaminase tests in chronic liver diseases, but it is especially suitable for the early differential diagnosis of viral hepatitis.

0554 ACTIVATION ANALYSIS OF HUMAN TISSUE SAMPLES: MANGANESE, COPPER, AND ZINC IN THE LIVER. *(Ger.)* Leonhardt, W.; Niese, S.; Jaross, W.; Hanefeld, M.; Schentke, K. U.; Renger, F. (Carl Gustav Carus Med. Acad., Dresden, Germany). *Dtsch. Z. Verdau. Stoffwechselkr.* 33(1):17-24, 1973.

Manganese, copper, and zinc contents were determined by radioactive irradiation in liver biopsies from rats, from 3 patients with hypertriglyceridemia, 4 patients with fatty liver, and 6 chronic alcoholics. Rat liver samples contained 6.1 ppm Mn, 15.1 ppm Cu, and 113 ppm Zn. The normal values in human liver were found to be 7.6 ppm Mn, 40 ppm Cu, and 181 ppm Zn. The corresponding values determined in fatty liver were 8.9 ppm Mn, 31 ppm Cu, and 135 Zn. Zn content was found to decrease with increasing triglyceride level in the liver. Correlation coefficients of 0.576, 0.599, and 0.448 were determined for the relation between the levels of Mn and Cu, Zn and Cu, and of Mn and Zn. Investigations of the Mn, Cu, and Zn levels in liver biopsies from alcoholics before and after tapering-off showed no clear tendency in the trace element concentrations.

0555 LIVER TRANSPLANTATION IN CHILDREN WITH BILIARY ATRESIA AND VASCULAR ANOMALIES. *(E.)* Lilly, J. R.; Starzl, T. E. (Univ. Colo. Med. Cent., Denver). *J. Pediatr. Surg.* 9(5):707-714, 1974.

Of 29 children having liver transplant for biliary atresia, 8 (27.5%) had associated vascular anomalies. In 3 of the latter a composite vascular malformation characterized by absent inferior vena cava, preduodenal portal vein and anomalous hepatic artery was also present, as well as visceral malformations such as intestinal malrotation, polysplenia or liver symmetry. The remaining 5 children had anomalous hepatic artery vasculature (4 cases) and malformation of the hepatic veins (1 case). All 3 infants with multiple vascular and visceral anomalies died shortly after liver homograft surgery. The visceral anomalies provide a special clue to the detection of this special group of infants with biliary atresia and multiple vascular malformation, and a radiologic study of the intestine with barium examination of the gastrointestinal tract should be a routine work-up for infants with jaundice. The polysplenia or liver symmetry seen at laparotomy should alert the surgeon to the possiblity of multiple vascular anomalies which make these patients a poor risk for successful liver transplant. A single variation of hepatic arterial or venous architecture is no contraindication for surgical correction and transplant.

0556 MAJOR HEPATIC RESECTION USING VASCULAR ISOLATION AND HYPOTHERMIC PERFUSION. *(E.)* Fortner, J. G.; Shiu, M. H.; Kinne, D. W.; Kim, D. K.; Castro, E. B.; Watson, R. C.; Howland, W. S.; Beattie, Jr., E. J. (Mem. Sloan-Kettering Cancer Cent. New York, N.Y.). *Ann. Surg.* 180(4):644-652, 1974.

A technique for *in vivo* isolation and asanguinous perfusion of the liver which permits hepatic resection to be performed under controlled conditions is described along with results from 29 major hepatic resections using the method. After isolation of the liver, the gastroduodenal artery is cannulated with a silastic catheter (1.1 mm internal diameter and 2.7 mm external diameter). The portal vein is cannulated either *via* a venotomy or *via* the branch of the lobe which is to be resected. These cannulae are kept patent with a slow infusion of Ringer's lactate at room temperature. The common bile duct is then occluded with a small Pott's clamp, and the liver is ready for hypothermic perfusion. The common hepatic artery and portal vein are clamped proximal to the cannulae. The infrahepatic vena cava and suprahepatic vena cava are separately occluded by vascular clamps, and a cavotomy is made immediately inferior to the liver for placement of a number 32 French catheter to drain the perfusate. Ringer's lactate solution, chilled to 4 C and with 5 mg/liter heparin, is passed through coiled tubing immersed in ice to the hepatic artery and portal vein. After traversing the liver, the solution exits through the hepatic veins into the vena cava. The liver is ready for asanguinous resection after obtaining adequate chilling of the parenchyma and checking for complete vascular isolation. Out of 29 patients undergoing the procedure, 26 survived. In 1 patient, isolation of the liver was incomplete and blood leaked into the system, resulting in hypovolemia, cardiac standstill, and death. Another patient died of pulmonary embolism 6 hr postoperative, while a 3rd patient died of hepatorenal syndrome about 4 weeks after surgery. Subphrenic abscess and wound infection were the most prominent complications. Resectability rate in the series of patients was 34%. Examples of surgical problems that can be dealt with

using the method are: resection of major hepatic veins flush with the vena cava, removal of a portion of the vena caval wall, excision of neoplastic involvement of liver tissue adjacent to vena cava, and neoplastic thrombosis of the portal vein.

0557 PRIMARY LIVER CANCER: A CLINICAL STUDY. *(Ger.)* Rostock, K. J.; Claus, R. (Med. Clin., Univ. Rostock, Germany). *Dtsch. Z. Verdau. Stoffwechselkr.* 33(4/5):193-198, 1973.

Fifty cases of primary carcinoma and 2 cases of hemangioendothelioma of the liver, observed between 1960 and 1972, are analyzed. Hepatocellular carcinoma in 33 cases, hepatocellular carcinoma with liver cirrhosis in 16 cases, cholangiocellular carcinoma in 17 cases, and those with cirrhosis in 4 cases were diagnosed. The patients included 32 males and 20 females, mostly aged 50-80 yr. Eleven cases of septal, 8 cases of postdystrophic, 1 case of biliary cirrhosis were found pathoanatomically, while clinical investigations revealed 12 cases of alcohol-induced cirrhosis, and 6 cases of hepatitic cirrhosis. Hypersplenism was detected in 11 out of 46 cases. Esophageal varices were found in cirrhotic patients only. Stenosis of the biliary duct due to tumor infiltration, hepatocellular and cholangiocellular carcinomas of the biliary duct were detected in 15, 9, and 8 cases, resp. Metastases were found most often in the liver, lungs, pleura, peritoneum, adrenal gland, skeleton, gallbladder, ovary, thyroid gland, kidney, brain, and lymph nodes. Ascites and pylethrombosis were found in 30 and 16 cases, resp. The serum bilirubin level, aspartate amino transferase activity, prothrombin and serum iron levels showed no significant changes, while the serum albumin, α_2- and β-globulin levels were pathological. The first symptom of the liver carcinoma was pain in 28 cases, jaundice in 6 cases, fever in 3 cases, and increase in the body circumference in 4 cases.

0558 RESULTS OF THE TREATMENT OF HEPATOCELLULAR CARCINOMA. *(Jpn.)* Kuroyanagi, Y.; Akaza, T. (Aichi Cancer Cent. Hosp., Japan). *Gan No Rinsho* 19(5):488-494, 1973.

The results of treatment of hepatocellular carcinoma were studied in follow-ups of 88 patients treated from 1964 through 1972. Group A patients were those seen from 1964 through 1968, before the development of the serum α-fetoprotein test, and whose diagnosis was based on hepatoscanning or selective angiography. Group B included the remainder of the patients. Treatment was by hepatic resection (5 cases), radiation combined with intra-arterial mitomycin C (22), single arterial mitomycin C (34), or a combination of venous mitomycin C, Tespamin, Endoxian and toyomycin (3). No treatment was given to 24 patients, who all died within 1 month. A single arterial injection of 20 mg mitomycin C was given to 3 of the 5 hepatic resection cases 1 week before surgery: 2 of these later developed serum metastasis to the lung and right pelvis, indicating that pre-operation treatment with mitomycin C was not effective. The 2 remaining surgical patients died of intestinal obstruction after 69 months and lung cancer after 58 months, resp.; however, no metastasis was observed to the remaining liver. In the radiation combined with Mitomycin C treatment, Betatron (cobalt 60) treatment was continued after the single arterial injection of 30 mg Mitomycin C. The effective radiation quantity was reached in 13 to 14 days of irradiation and was between 800 to 1500 r. At this quantity, the serum α-fetoprotein level was decreased to 86-88% of its initial level. The average survival period with this treatment was 10.7 months and 5.8 months for non-cirrhotic and cirrhotic hepatocellular carcinoma patients, resp. The single arterial mitomycin C treatment extended patient lives for only 4 months. The effectiveness of multi-drug venous injection, namely, 10 mg mitomycin C, 70 mg Tespamin, 3.5 mg Endoxian, and 3.5 mg toyomycin, was not observed clearly because of the complicated conditions of the patients, who all died within 2 months. Of the 88 original patients, the two treated by hepatic resection alone had the longest survival time, and had no evidence of further carcinomatous involvement.

0559 GLUTAMATE DEHYDROGENASE ACTIVITY RELATED TO HISTOPATHOLOGICAL GRADE OF HEPATOCELLULAR CARCINOMA IN MAN. *(E.)* Glazer, R. I.; Vogel, C. L.; Patel, I. R.; Anthony, P. P. (Dep. Pharmacol., Emory Univ., Atlanta, Ga.). *Cancer Res.* 34(11):2975-2978, 1974.

Twenty cm^3 blood was collected from each of 25 primary hepatocellular carcinoma patients (16 male, 9 female, 22-62 yr old). The sera was separated within 1 hr of collection and used for determinations of α-fetoprotein (AFP), protocollagen proline hydroxylase, alkaline phosphatase, and SGOT, and for serum bilirubin studies. Glutamate dehydrogenase was determined spectrophotometrically in biopsy samples of the primary tumor. Abnormally high activities of protocollagen proline hydroxylase, alkaline phosphatase, and SGOT were found in 53, 84, and 95% resp. of the patients with primary hepatocellular carcinoma. AFP levels ranged from 8 ng to 1.92 mg/ml; 75% of the patients had serum concentrations of AFP greater than 40 ng/ml. Bilirubin concentrations were elevated in only 21% of the patients. When the hepatic glutamate dehydrogenase activities were grouped according to the histological grade of the hepatocellular carcinoma, there was a suggestion of decreasing glutamate dehydrogenase activity with increasing anaplasia. Glutamate dehydrogenase activity of the hepatoma was significantly less than that obtained from adjacent normal or cirrhotic liver. Analyses of serum levels of AFP also suggested a correlation between the amount of AFP and the histopathological grade of the carcinoma.

0560 DIFFERENTIAL DIAGNOSIS OF NODULAR LESIONS IN THE LIVER OF MAN. *(Ger.)* Sterba, J.; Slais, J. (Parasit. Inst., Prague, Czechoslovakia). *Zentralbl. Allg. Pathol.* 118(2/3):124-131, 1974.

Systematic histological study of 122 human liver nodules obtained at autopsy and biopsy is described.

Solitary tumor metastases and specific granulomas were excluded from the investigation. Three different groups of liver nodules were distinguished: regressive and resorptive processes following different lesions (76 cases); parasitic granulomas and undetermined conditions (8 cases). The 1st group included serous cysts (35 cases), hyalinized angiomas, pseudoparasitic granulomas, implanted appendices epiploicae, and calcified focal liver necroses. Serous cysts showed focal dystrophic calcification and subcapsular localization. Pseudoparasitic granulomata from embolized plant parts *via* the portal vein due to rupture of the duodenum were localized in the portal region. Supracapsular pseudotubercles with partial necrotization and hyalinization due to plant seed was found in 1 case. Solitary calcified nodules due to coagulation necrosis was found in 4 cases. Special techniques and reconstruction were necessary for the diagnosis of parasitic granulomas caused by *Echinococcus granulosus, Linguatula serrata, Cysticercus tenuicollis, Enterobius vermicularis,* and *Capillaria hepatica,* the latter being detected for the 1st time in Europe in 7 cases. The identification of parasites, migrating to the liver from the intestine *via* the portal vein, is difficult due to resorption, necrosis, and calcification.

0561 ALPHA FETOPROTEIN: AN INDEX OF PROGRESSION OR REGRESSION OF HEPATOMA, AND A TARGET FOR IMMUNOTHERAPY. *(E.)* Parks, L. C.; Baer, A. N., Pollack, M.; Williams, G. M. (Johns Hopkins Univ. Sch. Med., Baltimore, Md.). *Ann. Surg.* 180 (4):599-605, 1974.

Studies of α-fetoprotein (AFP) producing hepatomas in humans and mice were performed to investigate possible correlations between serum AFP levels and tumor growth and to assess the ability of highly specific anti-AFP as a carrier for localizing diagnostic and possibly therapeutic amounts of radioactivity in hepatoma tissue. Measurements of serum AFP in patients with hepatomas, using a rabbit anti-human AFP as antisera, indicated a positive correlation between AFP titer and clinical status when the patient was used as his own control. Abnormal serum AFP levels in 2 patients with hepatomas resected for cure returned to normal over a period consistent with the 6-day half-life for circulating AFP. Recurrent disease was first detected by the reappearance of elevated AFP levels. Effective palliative anti-tumor therapy (5-fluorouracil, 12 mg/kg/week i.v.) did not cause any transient elevation of serum AFP levels which might be expected to accompany tumor cell destruction. Experiments with adult C57L/J mice bearing transplanted hepatomas (*via* s.c. placement) indicated that the sera of such animals positive for AFP at a titer of 1:64 could be cleared of circulating AFP by a single injection of 0.5 ml of anti-AFP. AFP titers fell to zero within 1 hr after injection. Although AFP serum activity resumed 10-18 hr post-injection, animals receiving repeated injections of 0.25 or 0.5 ml anit-AFP for 14 consecutive days after tumor inoculation remained clear until day 8. An initial attempt to utilize anti-AFP as a carrier for localizing selectively significant amounts of ^{131}I failed because the amount of anti-AFP antibody was low in relation to the amount of labeled but indifferent immunoglobulin G. However, anti-AFP antibody concentrated by precipitation with the specific antigen and dissociated from it by acidification and sephadex column purification did localize significantly in hepatoma (3 times the radioactivity of that found in hepatomas exposed to normal immunoglobulin G). The tumor:blood ratio of isotope activity was greater than 6:1 by the 6th post injection day, and therefore within the range suggested as necessary to localize therapeutic amounts of radioactivity.

0562 FOCAL HYPERFIXATION OF RADIOCOLLOID BY THE LIVER. *(E.)* Pasquier, J.; Dorta, T. (Cantonal Hosp., Fribourg, Switzerland). *J. Nucl. Med.* 15(8):725, 1974.

A liver scan in a 37-yr-old woman showed a "hot spot" which corresponded to a palpable mass in the epigastrium. Left liver-lobectomy, performed when angiography suggested a tumor, revealed a well-defined tumor in the left lobe of the liver. Histologic diagnosis was a hamartoma.

0563 STUDIES ON LIPID AND FATTY ACID COMPOSITION OF HUMAM HEPATOMA TISSUE. *(E.)* Araki, E.; Phillips, F.; Privett, O. S. (Hormel Inst., Univ. Minnesota, Austin). *Lipids* 9(9):707-712, 1974.

Studies were conducted on the lipid composition of normal liver and hepatoma obtained at autopsy from 8 human adults. Normal tissue was obtained from 3 patients who died of massive gastrointestinal bleeding. Total lipids were fractionated by sialic acid column chromatography into neutral lipid, glycolipid and phospholipid fractions which were further analyzed by thin layer chromatography. Constituent fatty acids were analyzed by gas liquid chromatography. Compared to normal liver tissue, hepatoma had a generally much higher, but more variable total lipid content (12.8-92.7 gm%) which was primarily due to increased neutral lipids (9.4-76.7 gm%). Although the glycolipid content of hepatomas was about the same as that of normal liver (0.6-0.8 gm%), phospholipid content was much lower (2.9-12.0 *vs* 11.7-13.7 gm%). Neither normal liver nor hepatoma tissue had any glyceryl ether diesters and both contained only traces of plasmalogens of phosphatidylcholine or phosphatidyl ethanolamine. Hepatomas contained higher levels of monoenoic acids and lower levels of linoleic and polyunsaturated fatty acids. Positional isomers of trienoic acids not normally present in liver tissue were detected in hepatoma tissue. The abnormalities in lipid composition observed in hepatoma tissue suggest that the normal regulatory processes of lipid metabolism are altered in a manner similar to those observed in tumors from animals.

0564 A RARE, HEREDITARY DISEASE WITH CHOLESTEROL ESTER STORAGE IN THE LIVER. *(Ger.)* Wolf, H.; Hug, G.; Michaelis, R.; Nolte, K. (Pediatr. Ward, Acad. Hosp., Kassel, Germany). *Helv. Paediatr. Acta* 29(2):105-118, 1974.

Follow-up observations for 10 years of cholesterol ester storage disease in a 17 yr-old girl are presented. Persistent type II hyperlipidemia and hepatomegaly were determined as the only abnormal clinical findings. Repeated liver biopsy specimens indicated excessive cholesterol esters both in liver cells and in the small intestinal mucosa. Acid lipase activity was deficient in the skin fibroblast cultures, while the other 7 lysosomal hydrolases showed normal activity. Serum lipid tests revealed a total cholesterol level of 400 mg/100 ml, a free cholesterol level of 150 mg/100 ml, a phosphatide level of 376 mg/100 ml, a neutral fat level of 353 mg/100 ml, a total ester fatty acid level of 750 mg/100 ml, and a total fat level of 1,325 mg/100 ml. Increased ester fatty acid, phosphatide, and cholesterol levels were demonstrated in both the father and mother of the patient. Additionally, the father had moderate hepatomegaly. One of the two siblings of the patient was found to have developed hepatomegaly, nearly pathological cholesterol level, and considerable fat storage in the astrocytes.

0565 DIVERSION OF THE HEPATIC ARTERIAL FLOW INTO THE PORTAL VEIN IN THE BUDD-CHIARI SYNDROME. *(E.)* Deutsch, V.; Adar, R.; Bogokowsky, H.; Itzchak, Y.; Mozes M. (Chaim Sheba Med. Centr., Tel-Hashomer, Israel). *Blood Vessels* 11(1/2):96-100, 1974.

A case of autopsy-proven primary Budd-Chiari syndrome with complete occlusion of all intrahepatic veins is described in which celiac angiography was successfully used to make a correct preoperative diagnosis. The patient, a 23-yr-old male, presented with hematemesis and melena. He was pale and slightly jaundiced. Spider angiomata were observed over the skin of the chest, and the abdomen was swollen with shifting dullness. The liver and spleen were not palpable. Hematologic laboratory tests showed anemia, hyperbilirubinemia (3.2 mg%), and mild abnormalities of liver function. Esophageal varices were suspected on barium swallow. Celiac and selective hepatic angiography demonstrated an enlarged liver, a slightly dilated hepatic artery, and intrahepatic branches with normal appearance and distribution. In the capillary phase of the selective hepatic injection, the intrahepatic portal vein branches were visualized. Long after the capillary phase, the splenic vein was not visualized. Superior mesenteric arterial injection revealed in the portovenous phase a dilated coronary vein with a rich collateral network. A diagnosis of hepatic vein occlusion was suspected on the basis of portal hypertension with extensive collaterals, direct filling of the intrahepatic portal vein from the hepatic artery, and an enlarged liver with normal vascular pattern. A portacaval shunt was constructed at surgery after which the portal pressure decreased from 44 to 28 cm H_2O. Postoperative hepatic encephalopathy appeared, and 2 episodes of gastrointestinal hemorrhage occurred. The patient died 10 days following the operation. Post mortem revealed a patent portacaval shunt, but with a thrombus partially occluding the portal vein 2 cm proximal to the anastomosis. With reticulum and collagen stains, the central hepatic veins were seen completely occluded by dense fibrosis. The larger hepatic veins were partially obliterated by intimal proliferation, some by organizing thrombus, while the major hepatic veins contained recent thrombus. The histological findings were indicative of primary Budd-Chiari syndrome (veno-occlusive disease). The direct evidence of shunting of blood from the hepatic artery into the portal vein, together with consideration of the angiographic pattern of the intrahepatic arterial branches, should be considered as very suggestive for the diagnosis of Budd-Chiari syndrome.

0566 ASSOCIATION OF REYE'S SYNDROME WITH VIRAL INFECTION. *(E.)* Linnermann, Jr., C. C.; Shea, L.; Kauffman, C. A.; Schiff, G. M.; Partin, J. C.; Schubert, W. K. (Univ. Cincinnati Coll. Med., Ohio). *Lancet* (7874):179-182, 1974.

From January to March 1974, 24 cases of Reye's syndrome (14 boys and 10 girls, mean age 7.7 yr) were diagnosed in Cincinnati, Ohio. In 16 cases the diagnosis was confirmed by liver biopsy. The epidemic coincided with an epidemic of influenza B, intermediate type. In 18 of the 23 cases (78%) in which viral studies were done, an acute viral infection was found in either the patient or a close contact with similar prodromal symptoms. Influenza-B infection was confirmed in 12 patients and 12 contacts, although influenza A, parainfluenza 1, adenovirus, and varicellazoster virus also occurred. In 4 cases, there was evidence of a mixed viral infection of patients or their contacts. Normal responses to phytohemagglutinin and specific viral antigen were obtained in cellular immunity studies performed on 6 patients. This is the first epidemic of Reye's syndrome clearly associated with viral infection.

0567 TREATMENT OF AMEBIC LIVER ABSCESS WITH EMETINE HYDROCHLORIDE, NIRIDAZOLE, AND METRONIDAZOLE. *(E.)* Datta, D. V.; Singh, S. A. K.; Chhuttani, P. N. (Postgrad. Inst. Med. Educ. Res., Chandigarh, India). *Am. J. Trop. Med. Hyg.* 23(4):586-589, 1974.

Three groups of 20 patients each with amebic liver abscesses were treated with 1 of the following antiamebic regimens: emetine hydrochloride (1 g i.m. for 10 days) with chloroquine diphosphate (250 mg 2 times/day for 20 days); niridazole (500 mg 3 times/day for 10 days); or metronidazole (400 mg 3 times/day for 10 days). Emetine + chloroquine resulted in 19 clinical cures. Clinical failure occurred in 1 of 2 patients who developed hypotension; this patient had associated rupture of the abcess into the peritoneum and had aspirated 800 ml of amebic pus after receiving only 1 g emetine. The other patient with hypotension recovered after emetine was reduced to 0.5 g for 3 days. Vomiting in 2 patients responded to antihistamines, and psychosis in 1 was relieved by withdrawing chloroquine. Of the 18 patients who completed the 10-day course of niridazole, 14 were cured. In 2 patients therapy was discontinued because of severe hallucinations, involuntary movements, and behavior changes. Recovery followed sub-

sequent treatment with phenobarbital and metronidazole. A 3rd patient died from hemorrhage into the abscess and a 4th developed peritonitis and paralytic ileus; the latter responded to emetine + chloroquine. Niridazole also caused severe vomiting (1 case) and hyperreflexia (2). Clinical failure occurred in 7 patients treated with metronidazole and in 4 of these *E. histolytica* was demonstrated in the hepatic aspirate at the end of therapy. One of the 7 patients developed severe hypotension and died after 3 days of treatment; autopsy revealed multiple abscesses teeming with *E. histolytica*. The other 6 patients responded to emetine-chloroquine therapy. Hallucinations in 1 patient and vomiting in 2 responded to phenobarbital. Information regarding absorption of different doses of metronidazole in amebic liver abscesses is lacking; higher dosages may prove more effective.

0568 VITAMIN B_{12} ABSORPTION IN CHRONIC LIVER DISEASE. *(Ger.)* Konrad, H.; Teichmann, W.; Kiencke, H.; Bielka, B.; Claus, R. (Med. Clin., Univ. Rostock, Germany). *Dtsch. Z. Verdau. Stoffwechselkr.* 33(4/5):179-183, 1973.

The radiolabeled vitamin B_{12} resorption was studied in 170 patients with different degrees and durations of chronic hepatic diseases, such as liver cirrhosis, chronic hepatitis, fatty liver, cardiac liver cirrhosis, and hepatocarcinoma by Schilling's vitamin B_{12}-urine excretion test. The vitamin B_{12} resorption showed an overall decrease in the patients, and pronounced pathological reduction in the vitamin B_{12} resorption was found in 30 patients (17.6%). While the vitamin B_{12} resorption was normal in 26 patients with chronic aggressive hepatitis and 17 patients with chronic persistent hepatitis, 11.8% of the 135 patients with cirrhosis, chronic hepatitis, and fatty liver showed pathological resorption values. The disturbance in the vitamin B_{12} resorption was most common in cardiac liver cirrhosis and hepatocarcinoma. Repeated tests with the addition of intrinsic factor (porcine powdered pylorus mucosa) showed normalization of the vitamin B_{12} resorption in most cases of cirrhosis, chronic hepatitis, and fatty liver. This effect was absent in certain patients, especially those with cardiac liver cirrhosis. About one third of the patients with pathological Schilling test had anemia. The anemia was mostly normochromic, and hyperchromic or hypochromic forms were found in a few cases.

0569 INTRACISTERNAL HYALIN IN HEPATOCYTES OF HUMAN LIVER BIOPSIES. *(E.)* Pfeifer, U.; Klinge, O. (Inst. Pathol., Univ. Wurzburg, Germany). *Virchows Arch. [Zellpathol.]* 16(2):141-155, 1974.

Further histologic studies have been conducted using liver biopsy material from 2 patients with cirrhosis and 1 with progressive liver damage to characterize one form of hepatocellular hyalin which accumulates inside the cisternae of the rough endoplasmic reticulum. The staining properties and ultrastructure of the material was not the same in the 3 cases. The PAS reaction was positive in only 1 specimen. The size of the hyalin inclusions varied considerably with the largest ones being 15 μ in one case but only 5-7 μ in the others. Ultrastructural studies showed that the inclusions were bounded by a single membrane similar to that of the endoplasmic reticulum. Attached ribosomes were common in two cases but rare in the 3rd. Occasional communications were noted between the inclusions and the endoplasmic reticulum. The substance within the inclusions was rather homogeneous in 2 cases and consisted of a pattern of irregularly arranged 400 Å tubules in 1 case. The inclusions were negative for acid phosphatase. The inclusions in 2 of the cases definitely did not represent accumulation of α_1-antitrypsin as has been noted in patients with α_1-antitrypsin deficiency. The accumulation could be a result of defective protein transport, of defective protein coupling or of synthesis of a defective protein.

0570 LIVER FINDINGS IN PRIMARY HYPERLIPOPROTEINEMIA (HLP). *(Ger.)* Renger, F.; Hanefeld, M.; Jaross, W.; Stotzner, H.; Kemmer, C.; Leonhardt, W.; Trubsbach, A.; Schentke, K. U. (Carl Gustav Carus Med. Acad., Dresden, Germany). *Dtsch. Z. Verdau. Stoffwechselkr.* 33(4/5):199-203, 1973.

Histological and biochemical liver findings in 6 cases of primary hyperlipoproteinemia type II and in 36 patients with primary hyperlipoproteinemia (HLP) types III-V are presented. The normal histological findings in hyperlipoproteinemia type II indicate that hypercholesterolemia *per se* fails to cause any serious morphological changes. Lipacidemia, hyperinsulinemia, and disturbances in the triglyceride metabolism were found in HLP types III-V. Steatosis hepatis was found in 33 cases (92%). Slight mesenchymal changes were detected despite massive fatty degeneration. Fine structural changes of the hepatocytes, mostly in the form of swelling with moderate to pronounced dilatation of the granular and agranular endoplasmic reticulum were found in electron microscopic investigations. The bile capillaries were often dilated, and small bile lipid occlusions were found in the surrounding plasma. Polymorphy of the mitochondria, giant mitochondria with paracrystalline occlusions, and amorphous substance, possible lipoproteins in the intercellular spaces were observed. Increased triglyceride concentrations were found in biopsies from patients with types III-V HLP, while biopsies from type II HLP were nearly fat-free. Significant relationship between the Broca index and the triglyceride content was established. The observed close relationship between the free fatty acid and liver triglyceride levels is indicative of triglyceride accumulation due to lipacidemia in patients with type III-V HLP. Significantly increased total bile acid level was measured in these patients compared with patients with type II HLP and with a control group. Abnormally high frequency of pathological thymol levels was established.

0571 THE EFFECT OF PHENOBARBITAL ON INTRAHEPATIC CHOLESTASIS OF PREGNANCY. *(E.)* Espinoza, J.; Barnafi, L.; Schnaidt, E. (Sch. Med., Catholic Univ., Santiago, Chile). *Am. J. Obstet. Gynecol.* 119(2):234-238, 1974.

Nine patients were treated for intrahepatic cholestasis of pregnancy. Sodium phenobarbital (2.5 mg/kg/day) administered for 4-16 days normalized the serum bilirubin levels in 7 patients whose initial concentration did not exceed 5 mg/100 ml. Two patients with pre-treatment bilirubin exceeding this level had a 50% decrease following therapy. When one of these two patients received phenobarbital in doses of 5 mg/kg/day her bilirubin level became normal. The effect of the drug may be due to enzyme-inducing action in the liver cell or to marked choluretic action.

0572 HIGH DENSITY LIPOPROTEINS IN CHOLESTASIS. *(E.)* Blomhoff, J. P. (Rikshosp., Oslo, Norway). *Scand. J. Gastroenterol.* 9(6):591-596, 1974.

High density lipoproteins from 2 patients (65-yr-old female and 49-yr-old male) with cholestasis and increased concentrations of plasma free cholesterol (82 and 306 mg/deciliter, resp.) and phospholipids (376 and 1000 mg/deciliter, resp.) but normal concentrations of cholesteryl esters (176 and 143 mg/deciliter, resp.) were studied and compared with results from previously described alterations in high density lipoproteins from patients with familial lecithin:cholesterol acyltransferase (LCAT) deficiency. Plasma LCAT activities, as measured by the methods of Stokke and Norum or Glomset and Wright, were near normal in the female patient (48 and 74 μM/liter/hr, resp.) and slightly decreased in the male (29 and 58 μM/liter/hr, resp.). High density lipoproteins were isolated by ultracentrifugation and gelfiltration on hydroxylapatite. Subsequent filtration through Sephadex G-200 resulted in the separation of high density lipoproteins from the 2 patients with cholestasis into 2 peaks, whereas normal high density lipoproteins were eluted in 1 peak. The major peak was eluted with or slightly after the void volume, a pattern similar to that previously described for patients with familial LCAT deficiency. All subfractions contained relatively more polar lipids (free cholesterol and phospholipids) than apolar (triglycerides and cholesteryl esters). The demonstration of heterogeneity of high density lipoproteins in cholestasis, irrespective of a normal capacity for plasma cholesterol esterification, supports the theory that the increase in plasma cholesterol and phospholipids in cholestasis may exceed the capacity of the LCAT enzyme.

0573 BILE SALT SULPHATES IN CHOLESTASIS. *(E.)* Stiehl, A. (Dep. Med., Univ. Heidelberg, Germany). *Eur. J. Clin. Invest.* 4(1):59-63, 1974.

Bile salt sulfates were chromatographically determined in serum and 24-hr urine samples of 40 patients with severe cholestasis (serum bilirubin greater than 10 mg% and serum alkaline phosphatase greater than 600 mμ/ml). The patients were equally divided into groups suffering from extrahepatic obstruction, hepatitis, cirrhosis, and metastases of the liver. The specific activities of cholate and cholate sulfates were determined without prior separation of secondary bile salts following i.v. injection of 25 μCi ^{14}C-cholate to a patient with extrahepatic obstruction. Sulfate esters in the urine of patients with cholestasis accounted for 46.9% of cholate, 62.6% of chenodeoxycholate, 63.2% of deoxycholate, 87.5% of lithocholate, and 55.8% of the total bile salts excreted. Monosulfates represented 76.9%, disulfates 21.3%, and trisulfates 1.8%. Bile salt renal clearance rates were not significantly different in patients with extrahepatic obstruction, hepatitis, cirrhosis, and metastases of the liver. Clearance of sulfated bile salts was 15 times greater than that for nonsulfated bile salts. In contrast to the urinalysis findings, only 5% of serum bile salts were sulfated. Serum concentrations of sulfated and nonsulfated bile salts did not significantly differ among the various groups of patients with cholestasis. Urinary excretion of mono- and disulfates of bile salts thus appears to represent an important excretory mechanism in patients with cholestatic liver disease.

0574 HEPATOCELLULAR CARCINOMA: RELATION TO ALCOHOL, HB ANTIGEN AND ALPHA-FETOPROTEIN. *(E.)* Dourdourekas, D.; Villa, F.; Szanto, P. B.; Ainis, H.; Steigmann, F. (Cook Cty. Hosp., Chicago, Ill.). *Gastroenterology* 67(4):789, 1974.

0575 PERITONEOSCOPIC OBSERVATION OF CHEMOTHERAPEUTIC EFFECTS IN A PATIENT WITH HEPATOCELLULAR CARCINOMA. *(E.)* Okazaki, N.; Hattori, N.; Arima, M.; Ono, T.; Kanda, Y.; Koyama, S. (Natl. Cancer Cent. Hosp., Tokyo, Japan). *Jpn. J. Clin. Oncol.* 4(1):77-80, 1974.

0576 THE VALUE OF LIVER BIOPSY. *(E.)* Schaffner, F. (Mt. Sinai Sch. Med., City Univ. New York, N.Y.). *Lab. Med.* 5(10):15-16, 20, 1974.

0577 LIVER BIOPSY: COMPARISON OF RISKS IN OUTPATIENTS AND IN-PATIENTS. *(E.)* McGill, D. B.; Ott, B. J.; Taylor, W. F. (Mayo Clin., Rochester, Minn.). *Gastroenterology* 67(4):814, 1974.

0578 ANTIBODY ACTIVATION OF MUTANT HUMAN FRUCTOSEDIPHOSPHATE ALDOLASE B IN LIVER EXTRACTS OF PATIENTS WITH HEREDITARY FRUCTOSE INTOLERANCE. *(E.)* Gitzelmann, R.; Steinmann, B.; Bally, C.; Lebherz, H. G. (Dep. Pediatr., Univ. Zurich, Switzerland). *Biochem. Biophys. Res. Commun.* 59(4):1270-1277, 1974.

0579 STUDIES ON LIVER ALDOLASES IN HEREDITARY FRUCTOSE INTOLERANCE. *(E.)* Schapira, F.; Hatzfeld, A.; Gregori, C. (Univ. Cochin Hosp. Cent., Paris, France). *Enzyme* 18(1/2):73-83, 1974.

0580 THE DIFFERENTIAL DIAGNOSIS OF NEONATAL HEPATITIS AND BILIARY ATRESIA. *(E.)* Campbell, D. P.; Poley, J. R.; Alaupovic, P.; Smith, E. I. (Univ. Oklahoma Health Sci. Cent., Oklahoma City). *J. Pediatr. Surg.* 9(5):699-705, 1974.

0581 NEONATAL AND RECURRENT CHOLESTASIS WITH LYMPHOEDEMA (NORWEGIAN TYPE HEREDITARY CHOLESTASIS). TWO NEW FAMILIES, WITH STUDIES ON BILE ACID METABOLISM AND ELECTRON MICROSCOPY. *(E.)* Aagenaes, O.; Normann, A. (Univ. Hosp. Rikshosp., Oslo, Norway). *Digestion* 10(4/5):332, 1974.

0582 DEFECTIVE METABOLISM OF THIHYDROXYCOPROSTANIC ACID (THCA) TO CHOLIC ACID (CA) IN TWO SIBLINGS WITH A FAMILIAL LIVER DISEASE: AN APPARENT DEFICIENCY OF 24-HYDROXYLASE. *(E.)* Hanson, R. F.; Isenberg, J. N.; Klein, P. D.; Williams, G. C.; Szezepanik, P.; Sharp, H. L. (Dep. Intern. Med., Univ. Minnesota, Minneapolis). *Gastroenterology* 67(4):796, 1974.

0583 DIFFERENTIAL RESPONSE OF SERUM BILIRUBIN AND SERUM BILE ACIDS IN CONSTITUTIONAL HEPATIC DYSFUNCTION (CHD). *(E.)* Robert, Y.; Paumgartner, G.; Preisig, R. (Dep. Clin. Pharmacol., Univ. Bern, Switzerland). *Digestion* 10(4/5):378, 1974.

0584 THE CEREBRAL COPPER-PROTEIN COMPLEXES IN HEPATO-LENTICULAR DEGENERATION STUDIED BY MEANS OF ELECTRON-SPIN-RESONANCE TECHNIQUE. *(E.)* Wender, M.; Adamczewska, Z.; Gruszczynska, U.; Dezor, A. (Inst. Phys., Polish Acad. Sci., Poznan). *Acta Neurol. Scand.* 50(6):812-815, 1974.

0585 IMMUNOLOGICAL STUDIES IN PATIENTS RECEIVING REPEATED ALLOGENEIC AND XENOGENEIC LIVER HEMOPERFUSIONS. *(E.)* Abouna, G. M.; Amemiya, H.; Andres, G.; Porter, K. A.; Hamilton, D. (Univ. Calgary Med. Sch., Canada). *Transplantation* 18(5):395-408, 1974.

0586 A STUDY OF LEVELS OF ORNITHINE CARBAMOYLTRANSFERASE IN 49 TUBERCULOSIS PATIENTS TREATED WITH RIFAMPICIN. *(E.)* Lengrand, J.; Kalachnikoff, P.; Martinez, B.; Rey, J. F.; Delmont, J. (Cimiez Hosp., Nice, France). *Digestion* 10(4/5):363, 1974.

0587 SYNTHESIS OF GLYCOSAMINOGLYCANS (GAG) AND COLLAGEN (C) BY HUMAN LIVER "FIBROBLASTS" IN TISSUE CULTURE. *(E.)* Galambos, J. T.; Hollingsworth, M.; Falek, A. (Emory Univ. Sch. Med., Atlanta, Ga.). *Gastroenterology* 67(4):794, 1974.

0588 STUDIES ON THE NATURE OF HYPERTRIGLYCERIDEMIA IN LIVER DISEASE. *(E.)* Linhart, P.; Seidel, D.; Kommerell, B. (Univ. Heidelberg Med. Clin., Germany). *Digestion* 10(4/5):365, 1974.

0589 ANAEROBIC BACTERIOLOGICAL STUDY OF THE HUMAN LIVER — WITH A CRITICAL REVIEW OF THE LITERATURE. *(E.)* Nielson, M. L.; Justesen, T.; Asnaes, S. (Rigshosp., Copenhagen, Denmark). *Scand. J. Gastroenterol.* 9(7):671-677, 1974.

0590 BILE SALT GLUCURONIDES IN MAN: IDENTIFICATION AND QUANTITATIVE ANALYSIS. *(E.)* Frohling, W.; Stiehl, A. (Med. Clin., Univ. Heidelberg, Germany). *Digestion* 10(4/5):318-319, 1974.

0591 BILE SALT ALKYL ETHERS: EVIDENCE FOR A NEW CLASS OF BILIARY LIPIDS IN HUMAN BILE. *(E.)* Hachey, D. L.; Szcepanik, P. A.; Watkins, J. B.; Tercyak, A. M.; Klein, P. D. (Argonne Natl. Lab., Ill.). *Gastroenterology* 67(4):795, 1974.

See also, 0055, 0156, 0176, 0177, 0194, 0212, 0329, 0339, 0340, 0342, 0367, 0368, 0369, 0370, 0371, 0372, 0373, 0374, 0375, 0376, 0381, 0419, 0593, 0666, 0679, 0688, 0694, 0716, 0738, 0778, 0782, 0797, 0798, 0799.

LIVER AND BILIARY TRACT

Hyperbilirubinemic States

0592 GILBERT'S SYNDROME IN PATIENTS WITH GALLBLADDER STONES. *(E.)* Peel, A. L. G.; Ritchie, H. D. (London Hosp., England). *Ann. R. Coll. Surg. Engl.* 55(4):184-189, 1974.

Gilbert's syndrome was diagnosed in 2 male patients (38 and 42 yr old) out of an unselected group of 247 cholelithiasis patients (63 males and 184 females). From these data it would appear that less than 1% of patients with gallbladder stones also have Gilbert's syndrome. However, when males alone are considered, the overall incidence was 3.2% with 95% confidence limits. Thus, about 1 in every 30 men with cholelithiasis may also have Gilbert's syndrome. It is suggested that the clinician be obligated to perform liver function tests on at least 2 occasions prior to cholecystectomy in male patients with cholelithiasis in an attempt to detect Gilbert's disease and avoid unnecessary bile duct surgery.

0593 ASSOCIATION BETWEEN HYPOURICAEMIA AND JAUNDICE. *(E.)* Schlosstein, L.; Kippen, I.; Bluestone, R.; Whitehouse, M. W.; Klinenberg, J. R. (Wadsworth VA Hosp. Cent., Los Angeles, Calif.). *Ann. Rheum. Dis.* 33(4):308-312, 1974.

Of 4148 patients screened during a 1-yr period, 22 (0.5%) had confirmed plasma urate levels below 2.2 mg/100 ml. Five of the 15 hypouricemic patients whose records were available for study had liver cirrhosis and jaundice. To determine whether the apparent association between hypouricemia and hyperbilirubinemia was a real one, the relation between

plasma urate and bilirubin was studied in 5 transiently jaundiced patients during their acute illness and later after complete or partial recovery. The mean serum bilirubin fell from 11.1 to 2.3 mg/100 ml as the patients recovered. This was accompanied by: (1) a rise in mean serum urate from 4.6 to 5.8 mg/100 ml; (2) a mean decrease in renal urate clearance of 3.2 ml/min; (3) a mean increase of plasma urate binding *in vitro* of 0.2 μg urate bound/mg albumin. Thus, the hypouricemia associated with hyperbilirubinemia may be effected in part by increased urate clearance. This increased urate clearance may be caused by bilirubin acting as a uricosuric agent. Further support for the relation between jaundice and impaired urate binding capacity was obtained from animal studies. In rats made icteric by bile duct ligation, urate binding to plasma proteins was completely abolished 20 hr after surgery.

0594 TOTAL SERUM BILE PIGMENT IN JAUNDICES OF VARIOUS ETIOLOGIES. *(Ger.)* Teichmann, W.; Jahn, H. (Med. Clin., Univ. Rostock, Germany). *Dtsch. Z. Verdau. Stoffwechselkr.* 33(4/5):163-166, 1973.

Results of the comparative determination of the total bile pigment and bilirubin levels in sera from 208 patients with viral hepatitis and 30 patients with extrahepatic obstructive jaundice are presented. The bilirubin level measured in patients with viral hepatitis ranged from 0.27 to 24.45 mg/100 ml, showing a difference of 0 to 2.75 mg/100 ml from the total bile pigment level. The serum bilirubin level in patients with obstructive jaundice ranged from 2.11 to 49.59 mg/100 ml, showing a difference of 0.05-5.60 mg/100 ml from the total bile pigment level. The difference is due to biliverdine and other pigments, which indicates that biliverdine is present in both viral hepatitis and obstructive jaundice. Therefore, the separate determination of the pigments is unsuitable for the differential diagnosis of viral hepatitis and obstructive jaundice. The absence of biliverdine in the serum in hemolytic jaundice indicates that conjugated bilirubin is oxidized much more easily than non-conjugated bilirubin.

0595 DIFFERENTIAL DIAGNOSIS OF JAUNDICE. *(Dut.)* Fevery, J. (St. Rafael Gen. Hosp., Louvain, Belgium). *Tijdschr. Geneesk.* 29(14):677-679, 1973.

0596 NEONATAL NONHEMOLYTIC JAUNDICE. *(E.)* Johnson, J. D. (Stanford Univ. Sch. Med., Calif.). *N. Engl. J. Med.* 292(4):194-197, 1975.

0597 BEHAVIOUR OF HEPATIC GLUCURONYL-TRANSFERASE ACTIVITY IN CHOLESTASIS AND OTHER DISEASES. *(E.)* Mazzanti, R.; Nassi, P.; Arcangeli, A.; Chiarantini, E.; Gentilini, P. (Dep. Intern. Med., Univ. Florence, Italy). *Digestion* 10(4/5):367-368, 1974.

0598 RECENT FINDINGS ON KERNICTERUS. *(It.)* De Toni, E.; Serra, G.; Lazzaroni, F. (L. Gaslini Inst. Pediatr., Univ. Genoa, Italy). *Rassegna Clin. Sci.* 49(4/5):119-124, 1973.

See also, 0124, 0166, 0167, 0377, 0571, 0676.

LIVER AND BILIARY TRACT

Nonviral Hepatitis and Hepatic Injury

0599 PELIOSIS HEPATIS ASSOCIATED WITH ANDROGENIC-ANABOLIC STEROID THERAPY: A SEVERE FORM OF HEPATIC INJURY. *(E.)* Bagheri, S. A.; Boyer, J. L. (Dep. Med., Univ. Chicago, Ill.). *Ann. Intern. Med.* 81(5):610-618, 1974.

Blood-filled cysts, charcteristic of peliosis hepatis, developed within hepatic parenchyma in 7 patients (5 men and 2 women, ages 20-67 yr) who were treated with androgenic-anabolic steroids for periods ranging from 2-27 months. Androgenic steroids were administered to 5 patients as hematopoietic agents to stimulate hypoplastic bone marrows, while the steroids were given as androgenic or anabolic drugs to 2 patients with malignancy. The agents used included testosterone enanthate, testosterone propionate, fluoxymesterone, methyltestosterone, norethandrolone, and oxymetholone. Although liver function was normal in all patients before therapy was started, all but 1 patient developed hepatomegaly or signs of hepatic toxicity following therapy. Two patients sustained intraperitoneal hemorrhages from the peliotic lesions, and 1 died from shock. Peliosis hepatis was a coincidental finding at autopsy in 3 of the patients who died from pulmonary alveolar proteinosis, Hodgkin's disease, and metastatic thymoma. Peliosis also seemed to contribute to the death of a patient in whom wide-spread breast cancer was found. Histologic examination suggested that the hemorrhagic cysts observed in the patients developed from focuses of hepatocellular necrosis. The findings indicate that peliosis hepatis can be a serious life-threatening complication of androgenic-anabolic steroid therapy in man.

0600 DISULFIRAM HYPERSENSITIVITY HEPATITIS. *(E.)* Keeffe, E. B.; Smith, F. W. (Portland VA Hosp., Oregon). *JAMA* 230(3):435-436, 1974.

A case of disulfiram-induced acute hepatitis is reported for a 44-yr-old male receiving the drug during treatment for chronic alcoholism and multiple

drug abuse. Disulfiram therapy (0.5 g daily for 5 days, then 0.25 g daily) had been initiated after 3 months of sobriety. After 10 days of therapy, the patient rapidly developed fatigue, malaise, and jaundice without fever. Disulfiram treatment was promptly stopped as was treatment with chlordiazepoxide hydrochloride and methylphenidate hydrochloride, both of which he had been taking for many months. The patient denied alcohol or other drug ingestion and knew of no exposure to hepatitis. The bilirubin value peaked at 6.9 mg/100 ml and the SGOT reading at 2040 U, with only a slight elevation in serum alkaline phosphatase level. Testing for hepatitis-associated antigen was negative. After 3 weeks of discontinuation of the drug, the patient felt well and liver function tests returned to normal. Ten days after resumption of disulfiram (0.25 g daily) therapy, the same biochemical and clinical sequelae returned. Different factors on this occasion included the presence of urticaria for 2 days and the absence of treatment with other medications. Again, the patient's condition improved rapidly after therapy with the drug was stopped. A liver biopsy specimen taken after the 2nd interruption in drug therapy was minimally abnormal, with mild cytoplasmic degeneration of hepatic cells, increased hepatocellular regeneration, and minimal focal histiocytic reaction. The patient was diagnosed as having probable disulfiram-induced hepatitis. The routine use of transaminase determinations before and after initiation of disulfiram therapy is suggested for detecting further cases of drug-induced hepatitis, particularly of the mild or anicteric variety.

0601 GRANULOMATOUS HEPATITIS DUE TO Strongyloides stercoralis. *(E.)* Poltera, A. A.; Katsimbura, N. (Dep. Pathol., Makerere Univ., Kampala, Uganda). *J. Pathol.* 113(4):241-246, 1974.

Autopsies were performed on 5 patients (2-45 yr old) who were infested with *Strongyloides stercoralis*. Serial sections of the available liver blocks were examined and special iron and reticulin stains were prepared. The *S. stercoralis* larvae had infiltrated the portal tracts and lobules in all 5 cases and in 1 case they were found in the portal vein branches. Cellular reaction was not always present. The number of larvae counted varied, the maximum being 13 in a 2 x 1 cm slide. Granuloma formation with or without larvae was observed in all cases, moderate diffuse fatty changes in the hepatocytes were noted in 4, cholestasis was present in 3 and several mitotic figures were observed in the liver cells of 2 of the patients. Four of the cases also had malaria, microfilaria or *S. mansoni* infestations. *Strongyloides stercoralis* should always be considered a possible cause in all cases of granulomatous hepatitis of obscure origin.

See also, 0145, 0150, 0378, 0444, 0783, 0796.

LIVER AND BILIARY TRACT

Acute Viral Hepatitis

0602 HEPATITIS — A VIRUS-INDUCED AUTOIMMUNE DISEASE. *(Ger.)* Berthold, H. (Med. Virol., Univ. Freiburg, Germany). *Verh. Dtsch. Ges. Inn. Med.* 79:637-639, 1973.

Adsorption tests with human fibroblasts and hepatitis serum, carried out by immunofluorescence, frequently revealed anticellular antibodies against human cells, e.g., of immunoglobulin G type. Changes caused by viral proliferation in the nuclear antigenicity can be also assumed to condition a change in the superficial antigenicity in hepatitis, in analogy to the SV 40 model. The immunologically altered cell is eliminated by the immune system as a foreign cell, in cooperation with the cellular immune reaction. This process of the immunological elimination of virus-infected cells is proposed to be termed as a virus-induced autoimmune process in connection with acute viral hepatitis.

0603 CELLULAR IMMUNE REACTIONS AGAINST THE HEPATITIS-ASSOCIATED ANTIGEN (HAA) AND THE HOMOLOGOUS LIVER-SPECIFIC ANTIGEN (HLP) IN ACUTE HAA-POSITIVE HEPATITIS. *(Ger.)* Knolle, J.; Meyer zum Buschenfelde, K. H.; Friedrichs, N.; Bolte, J. P. (Med. Clin. II, Univ. Mainz, Germany). *Verh. Dtsch. Ges. Inn. Med.* 79:639-641, 1973.

Cellular immune reactions against the hepatitis-associated antigen (HAA) and the homologous liver-specific antigen (HLP) were studied by Thor's modified leukocyte migration inhibition tests in acute HAA-positive hepatitis. Between the 3rd and 8th weeks of the disease, 46 cases of acute hepatitis were tested with HLP, and 51 with acute hepatitis with HAA-positive serum. In addition, 15 cases of cured hepatitis and 9 with chronic persistent hepatitis were investigated with HLP. Tests with HLP as an antigen revealed migration inhibition in 7 of 31 patients HAA-negative during the observation period. Similar findings were obtained for 3 out of 15 patients who remained HAA-positive. Inhibition was observed in 2 out of 15 patients with clinically cured hepatitis, and in 1 out of 9 patients with chronic persistent hepatitis. The findings indicate a possible virus-induced breakthrough of the tolerance against hepatic antigens. Acute hepatitis HAA-negative between the 3rd and 8th weeks of the disease showed inhibition of the migration in 20 of 33 cases, and stimulation in 5 of the 33 cases in the HAA test. Ten of the 18 persistent antigen carriers showed neither inhibition nor stimulation. Simultaneous inhibition of leukocyte migration in both the HLP and HAA tests was detected in 6 of the 51 cases, which indicates independent cellular immune reactions against HLP and HAA. Cellular immune reaction against HAA occurred most

frequently in cases in which the antigen disappeared during the observation period. It is concluded that cellular immune reactions against hepatic antigens are indicative of a virus-induced autoimmune reaction.

0604 RELATIONSHIPS BETWEEN INTESTINAL ALKALINE PHOSPHATASE ISOENZYME LEVELS IN BLOOD AND THE LENGTH OF THE COURSE OF VIRAL HEPATITIS. *(Fr.)* Gabrielle, L.; Leterrier, F.; Calba-Faguer, A.; Cristau, P.; Essioux, H.; Molinie, C.; Laverdant, C. (Res. Cent., Army Health Serv., Clamart, France). *Biol. Gastroenterol. (Paris)* 7(2):111-115, 1974.

The relation between the duration of viral hepatitis and serum levels of alkaline phosphatase isoenzyme of intestinal origin was studied. Based on the fact that 5 mM L-phenylalanine caused 10% inhibition of the bone and liver alkaline phosphatase isoenzymes and 60% inhibition of intestinal alkaline phosphatase, the following formula for "y", the serum level of intestinal enzyme, was derived: $y = 2(.9T - t)$ where T and t are the total activity of alkaline phosphatase before and after inhibition by L-phenylalanine, resp. Of 62 male hepatitis patients in their twenties, the 21 who had hepatitis for more than 2 months had significantly higher levels of intestinal alkaline phosphatase measured in the first 15 days of illness than the 41 who recovered within 2 months. There was no significant difference between these groups in the level of total or bone + liver alkaline phosphatase. In a control group of 60 healthy male patients in their twenties (30 of blood type O and 30 of blood type A), intestinal alkaline phosphatase was significantly higher in those with blood type O. This difference between the 2 blood types was not seen in the 44 hepatitis patients who were typed (23-O, 21-A).

0605 EPINEPHRINE-INDUCED GLYCOGENOLYSIS AND LYPOLYSIS IN INFECTIOUS HEPATITIS. II. NONESTERIFIED FATTY ACIDS AND THEIR RELATION TO IMPAIRED SUGAR UTILIZATION. *(Cz.)* Holik, K.; Rysanek, K.; Parak, S.; Prajsova, R. (Fac. Med., J. E. Purkyne Univ., Brno, Czechoslovakia). *Vnitr. Lek.* 19(1):49-54, 1973.

Fasting plasma nonesterified fatty acid (NEFA) levels were measured in blood taken from 10 patients with viral hepatitis and 14 normal controls 30, 60, 120, and 180 min after administration of 0.01 mg/kg epinephrine. Initial NEFA levels in patients with viral hepatitis were twice as high (0.480 mEq/liter) as those in controls (0.240 mEq/liter). Significant increases in plasma NEFA levels occurred in both controls and patients with viral hepatitis after administration of epinephrine, but increases were greater in patients with viral hepatitis than in controls. In both groups NEFA levels decreased rapidly from 60 min after epinephrine injections, and initial levels were reached after 120 min. These findings indicate that larger quantities of NEFA are mobilized from adipose tissue in patients with viral hepatitis and that this liberated NEFA is utilized rapidly. In a previous paper it was demonstrated that carbohydrate metabolism was impaired, insulin resistance increased, and the rate of gluconeogenesis increased in patients with viral hepatitis. These findings are similar to those of "fatty acid syndrome" described by Randle *et al.* in patients with diabetes, acromegaly, Cushing's syndrome, and starvation. Impairment of carbohydrate utilization causes this syndrome in liver disease.

0606 SUPPRESSION BY PREGNANCY AND BIRTH CONTROL PILLS (BCP) OF THE RISE IN SERUM GAMMA GLUTAMYL TRANSPEPTIDASE (GGT) IN VIRAL HEPATITIS. *(E.)* Combes, B.; Shore, G. M.; Cunningham, F. G.; Walker, F. B.; Shorey, J.; Ware, A. (Univ. Texas Southwest. Med. Sch., Dallas). *Gastroenterology* 67(4):A-784, 1974.

Serum gamma glutamyl transpeptidase (GGT) activity was compared in pregnant and nonpregnant women with viral hepatitis. Serum GGT was increased in the majority of women with viral hepatitis occurring in the first half of pregnancy. By contrast, in the second half of gestation GGT values were relatively depressed even though hepatitis was as severe. Mean GGT activity was lower, and abnormal values were less frequent in nonpregnant women with viral hepatitis who were taking birth control pills. It is suggested that estrogens and/or progestational compounds affect liver such that less GGT is released into blood with acute hepatocellular injury, and that GGT assays have impaired diagnostic value in persons exposed to estrogens and/or progestogens.

0607 HEPATIC CIRCULATION IN HEPATITIS A: A STUDY IN YOUNG MALES AT REST AND DURING AND AFTER SUPINE LEG EXERCISE. *(E.)* Lundbergh, P. (Roslagstull Hosp., Stockholm, Sweden). *Acta Med. Scand.* [*Suppl.*] (562):56pp, 1974.

The hepatic circulation of 10 male hepatitis A patients and 17 healthy male volunteers (age 20-30 yr) was studied by hepatic venous catheterization in the supine position at rest and during two 20-min periods of supine leg exercise and for 30 min thereafter. At rest in the supine position the patients had significantly higher values than the controls for total hepatic blood flow (2.21 liters/min *versus* 1.44 liters/min), for splanchnic oxygen uptake (79.7 ml/min *versus* 60.9 ml/min), for the difference between wedged and free hepatic venous pressure (2.9 mmHg *versus* 0.9 mmHg), and for estimated hepatic fat content (5.8% *versus* 3.6%). During exercise (10 patients and 13 controls), total hepatic blood flow decreased in both groups by about 0.8 liters/min. When the reduced hepatic blood flow during exercise was expressed as percent of the value at rest, the patients and the controls had the same flow reduction and increase in splanchnic flow resistance in relation to the increase in heart rate as a measure of the relative work load. Arteriohepatic venous oxygen difference during exercise was lower in patients than in controls. No negative effects of the exercise were seen in the 8 patients with values for serum bilirubin below 4 mg/100 ml but were noted in the two remaining patients with serum bilirubin values of 9.6 mg/100 ml and 5.8 mg/100 ml, resp. A probable ex-

planation of the high hepatic blood flow seen in the acute stage of hepatitis A might be that, because of the uneven distribution of the hepatic cellular changes, local circulation is unevenly impaired and anoxia develops in some areas with a resultant metabolic vasodilatation. It is concluded that the changes observed in the circulation during the acute stage of hepatitis A are attributable to the disease, that they are reversible, and that strenuous physical exercise should be avoided until the serum bilirubin is below 4 mg/100 ml.

0608 LONG TERM FOLLOW-UP STUDIES OF PATIENTS SURVIVING FULMINANT VIRAL HEPATITIS. *(E.)* Karvountzis, G. G.; Redeker, A. G.; Peters, R. L. (Univ. South. California Sch. Med., Los Angeles). *Gastroenterology* 67(5):870-877, 1974.

Long term follow-up studies (3 to 46 months) were conducted in 22 patients (15 to 34 yr) who had survived acute viral hepatitis with coma. The epidemiological background included the self-injection of drugs in 13 cases, no obvious source for the infection in 8 patients, and blood transfusion 3 months before jaundice in 1 patient. Complete clinical and biochemical resolution of the hepatitis was observed in all patients within 45 to 75 days after the onset of the acute hepatitis. The only identifiable sequela of the fulminant episode was the persistence of hepatitis B antigen (HB Ag) in 1 of 16 initially HB Ag-positive patients. Three patients suffered a second attack of acute viral hepatitis 4 to 10 months after recovery from the fulminant episode. All 3 patients had continued the use of illicit drugs. Mild unresolved hepatitis followed the second episode in 1 patient. Follow-up liver biopsy in 13 patients (2-37 months after the fulminant episode) showed no liver disease related to the fulminant hepatitis except for the mild unresolved hepatitis in the HB Ag carrier. It appears that patients who survive fulminant viral hepatitis rarely develop chronic liver disease, in spite of the extreme degrees of hepatic necrosis occurring during the acute illness.

0609 HEPATITIS B INFECTION: A POINT-PREVALENCE STUDY IN 15 US HEMODIALYSIS CENTERS. *(E.)* Szmuness, W.; Prince, A. M.; Grady, G. F.; Mann, M. K.; Levine, R. W.; Friedman, E. A.; Jacobs, M. J.; Josephson, A.; Ribot, S.; Shapiro, F. L.; Stenzel, K. H.; Suki, W. N.; Vyas, G. (New York Blood Cent., N.Y.). *JAMA* 227(8):901-906, 1974.

Five hundred eighty-three patients and 451 medical personnel of 15 hemodialysis centers in six states, representing a broad cross-section of facilities during 1972-1973, were surveyed for hepatitis B infections. In addition to the patients and medical personnel, serum specimens were obtained from 156 family contacts of 68 dialysis patients; 247 family contacts of 107 hepatitis B antigen (HB Ag)-negative volunteer blood donors served as controls. Serum samples were tested for HB antigen by agar gel diffusion and radioimmunoassay; for hepatitis B antibody (HB Ab) by passive hemagglutination; and for SGPT activity by the kinetic spectrophotometric technique. Epidemiologic data on patients, staff, and family contacts were gathered by personal interview and medical records. HB Ag was detected in 16.8% of the patients and in 2.4% of the medical staff; HB Ab was detected in 34.0% and 31.3%, resp. The cumulative prevalence of HB Ag and HB Ab varied widely among the centers. The prevalence of HB Ag and HB Ab in patients was related to duration of dialysis treatment, but not to blood transfusions. Significant differences in prevalence of serologic indicators of hepatitis B infection among various staff categories were not observed. Sixty-one % of the family contacts of dialysis patients with a history of hepatitis B infection were found to have HB Ag or HB Ab.

0610 ARTHRITIS IN ACUTE HEPATITIS AND CHRONIC ACTIVE HEPATITIS: PATHOLOGY OF THE SYNOVIAL MEMBRANE WITH EVIDENCE FOR THE PRESENCE OF AUSTRALIA ANTIGEN IN SYNOVIAL MEMBRANES. *(E.)* Schumacher, H. R.; Gall, E. P. (VA Hosp., Philadelphia, Pa.). *Am. J. Med.* 57(4):655-664, 1974.

Arthritis seen in the prodrome of acute viral hepatitis and as an early manifestation of chronic active hepatitis was studied in two patients using light and electron microscopic findings of needle synovial biopsy specimens. Both patients had very low levels of serum complement and circulating Australia antigen, suggesting the possible mechanism of immune complex deposition in some tissue injury. The synovial effusion in acute hepatitis was inflammatory, but neither synovial membrane showed more than rare inflammatory cells. Evidence for Australia antigen in the synovium was obtained by direct immunofluorescence and by electron microscopic identification of 200 to 250 Å and 400 to 600 Å particles in vessel endothelium, synovial lining cells and other deep synovial cells. Cell injury in synovial cells containing virus-like particles suggests that a direct virus effect on the synovium may be the mechanism for the production of arthritis.

0611 VIRAL HEPATITIS B AND PERIARTERITIS NODOSA. *(Fr.)* Trepo, C.; Thivolet, J.; Girard, M. (Red Cross Hosp., Lyon, France). *J. Med. Lyon* 54(1240):17-26, 1973.

Various reported observations indicate that the Australia antigen of hepatitis B (HB Ag) is associated with periarteritis nodosa in 1 case out of 3, but the antigen appears to be absent in other cases of vascularitis. The authors observed 24 cases of HB Ag associated with periarteritis nodosa. The antigen appeared early in the course of the disease in most instances, and in all but 3 of the cases the antigen persisted for as much as 2 years. The relation between the hepatitis virus and the disease is confirmed by the appearance of viral hepatitis at the beginning of the disease in 70% of the cases. In most cases the transaminases normalized in spite of the persistance of the antigen, especially when treated with cortisone. Thus the group of patients studied was distinguished clinically by the frequency of hepatic involvement and rarity of pulmonary

symptoms, and pathologically by the presence of HB Ag and absence of antinuclear antibodies and rheumatoid factors. These facts and the existence of contamination by the hepatitis virus preceding appearance of any symptoms in 15 cases shows that this is not a matter of a secondary infection favored by an immunological deficiency. Rather, it seems likely that the hepatitis virus plays a role in the etiology of periarteritis nodosa, a conclusion which is supported by some previous clinical, epidemiological, and animal studies. The hepatitis inaugurates the course of disease, but it may recede while the disease continues to evolve; this suggests that the engendering mechanisms of the 2 diseases are quite distinct. The factors which favor the development of periarteritis nodosa pursuant to hepatitis are unknown. Furthermore, since neither the antigen nor the antibody of HB Ag can be detected in some cases of periarteritis nodosa, it seems probable that chronic infection of hepatitis virus B constitutes only one of several etiologies of periarteritis nodosa.

0612 JAUNDICE AND HEPATITIS B ANTIGEN/ANTIBODY IN HEMOPHILIA. *(E.)* Lewis, J. H.; Maxwell, N. G.; Brandon, J. M. (Dep. Med., Univ. Pittsburgh, Pa.). *Transfusion* 14(3):203-211, 1974.

The incidence of hepatitis as well as the occurrence of jaundice and of hepatitis B antigen/antibody in over 1000 stored and fresh serum samples were studied in 375 hemophiliacs. Patients at highest risk were those who had received fractions prepared from pooled plasma (22%). Concentrates of factor IX were associated with hepatitis about 3 times as often as were factor VIII fractions. Acute phase serum was available from 12 of 14 hepatitis patients treated with factor VIII and 8 of 10 treated with factor IX concentrates. Of these, 2 of the samples from factor VIII deficient patients and 7 of the sera from factor IX deficient patients were positive for hepatitis B antigen. Antibody was detected in 6 factor VIII and 1 factor IX deficient sera. Multiple serum samples were available from 89 patients without overt hepatitis but who had received fraction therapy. Hepatitis B antigens were detected in 1 hemophiliac and 1 afibrinogenemic. Antibody was found in 24% of the factor VIII and 17% of the factor IX deficient patients. Positive antibody titers usually appeared following therapy with plasma fractions and decreased or disappeared during clinically quiescent periods. Of the 113 patients who received plasma fraction therapy, 66 (58%) did not develop overt hepatitis or circulating hepatitis B antigen/antibody.

0613 CELL-MEDIATED IMMUNITY TO HEPATITIS B ANTIGEN AND TO LIVER-SPECIFIC ANTIGEN IN ACUTE VIRAL HEPATITIS TYPE B. *(E.)* Lee, W. M.; Mitchell, C. G.; Reed, W. D.; Vahrman, J.; Zuckerman, A. J.; Eddleston, A. L. W. F.; Williams, R. (Kings Coll. Hosp. Med. Sch., London). *Gastroenterology* 67(4): 807, 1974.

0614 IMMUNOGLOBULINS IN ACUTE HEPATITIS. *(Ger.)* Stiess, G.; Berthold, H. (Dep. Res. Med. Virol., Univ. Freiburg, Germany). *Verh. Dtsch. Ges. Inn. Med.* 79:635-637, 1973.

0615 LYMPHOCYTE STIMULATION BY SUBTYPES OF HB_SAG. *(E.)* de Gast, G. C.; Houwen, B.; Que, G. S.; Nieweg, H. O. (Dep. Med., Univ. Groningen, Netherlands). *Digestion* 10(4/5):327, 1974.

0616 PLASMA LIPIDS AND LIPOPROTEIN CHANGES IN ACUTE VIRAL HEPATITIS. *(E.)* Pearson, A. J. G.; Pyrovalakis, J.; McIntyre, N. (R. Free Hosp., London, England). *Digestion* 10(4/5):322, 1974.

0617 A STUDY ON HEPATIC ENZYME INDUCTION DURING HUMAN ACUTE VIRAL HEPATITIS. *(E.)* Carrella, M.; Budillon, G.; Mazzacca, G.; De Ritis, F. (Inst. Clin. Med., Univ. Naples, Italy). *Digestion* 10(4/5): 341, 1974.

0618 CELLULAR IMMUNE REACTIONS IN THE COURSE OF VIRUS HEPATITIS. *(E.)* Sodomann, C. P.; Havemann, K.; Martini, G. A. (Med. Clin., Univ. Marburg/Lahn, Germany). *Digestion* 10(4/5):328-329, 1974.

0619 INTRAHEPATIC LOCALIZATION OF HEPATITIS B ANTIGEN (HBAg) BY IMMUNOFLUORESCENCE IN 275 LIVER BIOPSIES. *(E.)* Sanchez-Cuenca, J. M.; Roderigo-Moreno, M.; Rayon, M.; Gonzales-Molina, A.; Sarrion, J.; Marty, M. L.; Berenguer, J.; Baguena, J. (La Fe City Sanit. Soc. Secur., Valencia, Spain). *Digestion* 10(4/5):379-380, 1974.

0620 IMMUNOFLUORESCENCE AND ELECTRON MICROSCOPIC STUDY OF THE HEPATITIS B "CORE" ANTIGEN IN THE LIVER. *(E.)* Hadziyannis, H.; Gerber, M. A. (Athens Univ. Hippokration Gen. Hosp., Greece). *Gastroenterology* 67(4):795, 1974.

0621 BLEEDING DISORDER IN AN INFANT ASSOCIATED WITH ANICTERIC HEPATITIS. ACQUIRED PROTHROMBIN DEFICIENCY. *(E.)* Poley, J. R.; Humphrey, G. B. (Child. Mem. Hosp., Oklahoma City, Okla.). *Clin. Pediatr.* 13(12):1045-1047, 1974.

0622 SUBACUTE HEPATIC NECROSIS IN ACUTE HEPATITIS. *(E.)* Ware, A. J.; Eigenbrodt, E.; Combes, B. (Univ. Texas Southwest. Med. Sch., Dallas). *Gastroenterology* 67(4):835, 1974.

0623 HEPATITIS B IN PLASMA FRACTIONATION WORKERS. A SEROEPIDEMIOLOGIC STUDY. *(E.)* Taylor, J. S.; Shmunes, E.; Holmes, A. W. (Public Health Serv., Cincinnati, Ohio). *JAMA* 230(6):850-853, 1974.

0624 HEPATITIS-B IN AN ONCOLOGY UNIT. *(E.)* Wands, Wands, J. R.; Walker, J. A.; Davis, T. T.; Owens, A. H.; Carpenter, C. C. J. (Johns Hopkins Univ. Sch. Med., Baltimore, Md.). *Gastroenterology* 67(4): 834, 1974.

0625 LIVER DISEASE IN CHRONIC CARRIERS OF HEPATITIS B ANTIGEN (HBAg): HISTOLOGICAL FINDINGS IN 29 HEALTHY BLOOD DONORS. *(E.)* Villeneuve, J. P.; Cote, J.; Richer, G.; Guevin, R.; Marleau, D.; Joly, J. G.; Viallet, A. (St. Luke Hosp., Montreal, Canada). *Gastroenterology* 67(4):833, 1974.

0626 INTRAFAMILIAL SPREAD OF THE HEPATITIS B VIRUS. *(E.)* Hadziyannis, S. (Athens Univ. Med. Sch., Greece). *Digestion* 10(4/5):353, 1974.

0627 IMMUNOLOGICAL STUDIES ON LIVER DISEASE IN THE MONGOLIAN PEOPLE'S REPUBLIC. *(E.)* Akhmeteli, M. A.; Vasil'eva, V. I.; Dordzgotov, N.; Namdava, N.; Cernov, K. S.; Sipaceva, N. B. (World Health Organ., Geneva, Switzerland). *Bull. WHO* 50(5):407-411, 1974.

0628 FAMILIAL CLUSTERING OF HEPATITIS B SURFACE ANTIGEN AMONG PANAMANIAN INDIANS. *(E.)* Reeves, W. C.; Peters, C. J.; Moon, T. E.; Purcell, R. H. (Gorgas Mem. Inst. Trop. Prev. Med., Balboa Heights, Canal Zone). *J. Infect. Dis.* 131(1):67-70, 1975.

0629 ACUTE AND CHRONIC POST TRANSFUSION HEPATITIS. *(E.)* Koretz, R. L.; Damus, K.; Ritman, S.; Arthur, M.; Kash, P.; Golub, L.; Rebhun, D.; Gitnick, G. L. (Univ. California, Los Angeles). *Gastroenterology* 67(4):804, 1974.

0630 CURRENT PROBLEMS OF VIRAL HEPATITIS. *(It.)* Ricci, G. (Clin. Infect. Dis. I, Univ. Rome, Italy). *Rassegna Clin. Sci.* 49(3):73, 76, 1973.

0631 HEPATITIS. *(Fl.)* De Groote, J. (St. Rafael Gen. Hosp., Louvain, Belgium). *Tijdschr. Geneesk.* 29(14):680-686, 1973.

See also, 0379, 0594, 0636, 0684.

LIVER AND BILIARY TRACT

Chronic Hepatitis

0632 CHRONIC SEPTAL HEPATITIS. *(E.)* Gerber, M. A.; Vernace, S. (Mt. Sinai Sch. Med., City Univ. New York, N.Y.). *Virchows Arch.* [*Pathol. Anat.*] 363(4):303-309, 1974.

A type of chronic hepatitis designated as chronic septal hepatitis was distinguished morphologically from chronic aggressive and chronic persistent hepatitis in 18 of 200 biopsy specimens from patients with chronic hepatitis. The 18 specimens showed chronic portal inflammation without erosion of the limiting plate but with formation of fibrous septa. The portal tracts were moderately enlarged and infiltrated predominantly by lymphocytes mixed with plasma cells and macrophages. The inflammatory cells did not extend into the adjacent liver parenchyma, and the limiting plate was intact. There was little bile ductular proliferation and no ductal destruction. Fibrous septa of various lengths extended from the portal tracts into the parenchyma but only rarely reached the central veins or adjacent portal tracts. Clinically, many patients were asymptomatic despite hepatomegaly and elevated serum transaminase activities, while immune markers were usually absent from the serum. Chronic septal hepatitis was often seen after corticosteroid therapy of chronic aggressive hepatitis, though it also occurred *de novo*. Transition to cirrhosis was not observed during follow-ups (2 months to 5 yr) except in 1 case with high serum γ-globulin. Until the etiology of different types of chronic hepatitis is firmly established, classification will have to be based on clinical manifestations or morphologic criteria.

0633 CHRONIC LOBULAR HEPATITIS. *(Fr.)* Gardiol, D.; Rivier, E. (Cantonal Hosp., Lausanne, Switzerland). *Schweiz. Med. Wochenschr.* 104(41): 1450-1453, 1974.

Out of 9600 reviewed liver biopsies, 21 cases were found to conform to the recently fixed description of chronic lobular hepatitis: a process that continues for at least 3 months and presents a clinical picture of acute viral hepatitis, with disseminated hepatocyte necrosis in the hepatic lobe. The 21 cases were submitted to a systematic histological analysis. The most significant histologic sign for the diagnosis is the disseminated hepatocyte necrosis. Associated with this must be a diffuse kupfferian reaction and portal inflammatory infiltration. The criterion of chronicity is not demonstrated by any histologic signs, but 85% of the biopsies were done between the 4th and 6th months in the evolution of the disease. In 10 cases, it was possible to follow the evolution of the disease through more than one biopsy. It was found that after an interval of some months the lesions tended to recede, and in all but one case, biopsies taken more than a year after the initial episode of the disease showed more complete regression of the necrosis process. Chronic lobular hepatitis is distinguished from protracted hepatitis by more marked portal fibrosis and collagenization of the reticuline fibers, by less frequent cholestasis, and by less diversified hepatic involvement. It is distinguished from classic chronic hepatitis by greater hepatocyte necrosis. However, the term used to designate the disease is not satisfactory.

0634 CHRONIC FOCALIZED GLOMERULITIS AND CHRONIC PERSISTENT HEPATITIS FOLLOWING KIDNEY

TRANSPLANTATION: DETECTION OF HEPATITIS-B- AND EPSTEIN-BARR VIRUS ANTIGENS. *(Ger.)* Sonnabend, W.; Kistler, G. S.; Thiel, G.; Zollinger, H. U.; Bianchi, L.; Enderlin, F.; Gudat, F.; Krech, U. (Inst. Pathol., Univ. Basel, Switzerland). *Schweiz. Med. Wochenschr.* 104(35):1205-1217, 1974.

A kidney transplant recipient with primary chronic glomerulonephritis, investigated after an unclear rejection episode, was found to have chronic persistent B hepatitis and a chronic Epstein-Barr virus (EBV) infection. Hepatitis B antigen and EBV antigen were detected by immunofluorescence in the glomerular basal membranes of the transplant. The glomerula displayed intramembranous and subepithelial electron-dense deposits containing numerous small clusters of round particles measuring 20-30 nm and 100 nm, resp. The small particles are assumed to represent complexed HB_s-Ag, whereas the large particles are believed to represent nucleocapsids of EBV. Three later biopsies showed the development of a finally diffuse membrano-proliferative glomerulonephritis. Several episodes of increased proteinuria, which were paralleled by an increase in EVB antibody titer, point to a possible causal relationship between transient deterioration of the transplant function and the activation of the viral infection. The data thus support the findings of other investigators who postulate that even mild viral, bacterial and fungal infections may trigger the rejection of a renal allograft. The presence of HB Ag in serum samples taken between the first and 4th weeks post operation, a transient disappearance, and subsequent appearance indicate the possible presence of hepatitis B virus in the blood used for transfusion, and endogenous synthesis of HB Ag during the later course of the follow-up study.

0635 ASYMPTOMATIC LIVER DISEASE IN HEPATITIS B ANTIGEN CARRIERS. *(E.)* Woolf, I. L.; Boyes, B. E.; Jones, D. M.; Whittaker, J. S.; Tapp, F.; MacSween, R. N. M.; Renton, P. H.; Stratton, F.; Dymock, I. W. (Univ. Hosp. South Manchester, England). *J. Clin. Pathol.* 27(5):348-352, 1974.

Thirty-four healthy blood donors (25 male and 9 female, mean age of 33.6 yr), found to be persistent hepatitis B antigen carriers, were investigated by means of serial liver function tests, BSP retention, and liver biopsy. A history of possible exposure to the antigen was elicited in 19 of the donors. The only clinical abnormality was minor hepatomegaly which was present in 3 donors. Histological abnormalities were observed in 31 of the donors and included 1 case of cirrhosis, 3 cases of chronic aggressive hepatitis, and 11 cases of chronic persistent hepatitis. Focal areas of necrosis in the liver parenchyma were observed in 13 biopsies. Abnormal liver function (SGOT over 40 Karmen U or serum globulin greater than 3.5 g/100 ml) was detected in 18 of the donors, including 7 of 11 donors with chronic persistent hepatitis, 1 with cirrhosis, and all 3 with chronic aggressive hepatitis. Of the 13 donors with focal necrosis, 7 showed abnormalities of liver function. None of the 3 donors with normal histology showed any liver function abnormalities. BSP retention tests were abnormal in 15 cases, with greater than 6% retention of a standard 5 mg/kg dose at 45 min. These included the donor with cirrhosis, 6 donors with chronic persistent hepatitis, and all 3 donors with chronic aggressive hepatitis. Only 2 of the donors with focal necrosis and none of the 3 with normal histology showed abnormal BSP retention. A BSP retention of 7-11% was found in 5 of 6 donors with chronic persistent hepatitis, while all 3 donors with chronic aggressive hepatitis had a BSP retention of more than 11%. The severity of the histological changes was related neither to the titer of the antigen in the serum nor to the presence of autoantibodies.

0636 IMMUNOGLOBULIN AND AUTOANTIBODY RESPONSE IN ACUTE AND CHRONIC LIVER DISEASE. *(E.)* Vittal, S. B. V.; Dourdourekas, D.; Shobassy, N.; Ainis, H.; Clowdus, B. F.; Steigmann, F. (Hektoen Inst. Med. Res., Cook Cty. Hosp., Chicago, Ill.). *Am. J. Med.* 57(4):546-550, 1974.

Serum immunoglobulin and nonorgan-specific autoimmune responses (autoantibody response) were studied in 269 patients with a variety of acute and chronic liver diseases. About 75% of the patients with either hepatitis B antigen (HB Ag)-positive (40) or HB Ag-negative acute viral hepatitis (40) showed a mild increase in total globulin, γ-globulin, and immunoglobulin G (IgG) levels and a moderate elevation in IgM levels during the first 2 weeks of illness. These values began to subside by the 8th week and were completely normal by the 12th week in association with clinical improvement and return of SGOT values to normal. Serum complement (C_3) levels were low in 20% of the patients with acute viral hepatitis during the first 2 weeks of illness and returned to normal thereafter. Among the patients with acute viral hepatitis, 42.5% had smooth muscle antibody, 20.5% had mitochondrial antibody, 10% rheumatoid factor, and none had antinuclear antibody. These nonorgan-specific autoimmune markers were only transiently present (mean 2 weeks) and became undetectable as the patient's condition improved. There was no difference in these immunologic responses between HB Ag-positive or negative groups. Total globulin, γ-globulin, and IgG levels were above the range of normal in patients with chronic active liver disease (13), alcoholic cirrhosis (82), and in cirrhotic patients with hepatomas (5). Serum IgA levels were increased only in patients with alcoholic liver disease and were more evident in patients with alcoholic hepatitis (7) and cirrhosis. Elevated Ig levels were seen in HB Ag-positive and negative chronic active liver disease, alcoholic hepatitis, and alcoholic cirrhosis. IgM levels were normal in primary (10) and secondary (14) malignant liver disease. HB Ag-positive blood donors with asymptomatic mild liver disease had normal immunochemistry values. Serum complement levels were lower than normal in 50% of patients with chronic liver disease, with or without HB Ag, while C_3 values were normal in the other varieties of chronic liver disease. Smooth muscle antibody was present in 35% of the patients with HB Ag-positive chronic active liver disease (9) and in 20% of the patients

with HB Ag-negative chronic active liver disease (4). Antinuclear antibody was present in only 1 of 4 patients with HB Ag-negative chronic active liver disease. Mitochondrial antibody was not seen in any of the patients with chronic active liver disease. Of 10 patients with cirrhosis and hepatoma, 3 had HB Ag, 4 had mitochondrial antibody, 6 had smooth muscle antibody, and 4 had α-fetoprotein. The differentiation of type A hepatitis from type B hepatitis is concluded as impossible from the determination of serum IgM levels. Chronic active liver disease and cirrhosis appear to be associated with a potent stimulus to the proliferation of antibody-synthesizing cells resulting in hypergammaglobulinemia.

0637 AUTOIMMUNE REACTION OF THE "CHISIU TYPE" IN CHRONIC DISEASES OF THE LIVER. *(E.)* Costica, I. I.; Dumitru, M.; Benea, F. (23 August Hosp., Bucharest, Rumania). *Roum. Med. Rev.* 18(1):27-31, 1974.

Twenty-eight patients (9 females and 19 males, 20-69 yr old) suffering from chronic diseases of the liver were studied for the presence of antibile autoantibodies. Of the 28 patients, 18 had chronic hepatitis, 8 cirrhosis of the liver, and 2 cancer. The presence of Australia antigen and of antibile autoantibodies was determined by double immunodiffusion in agarose gel. The antigenic type and immunoglobulin class were determined by immunoelectrophoresis. Antibile autoantibodies were detected in 15/28 patients (53.56%) and Australia antigen in 6 cases (21.4%). The concomitant presence of Australia antigen and autoantibodies was found in only 3 patients. There was a statistically significant difference in alkaline phosphatase and bilirubin values between the patients with and without autoantibodies, and in 6/15 positive cases, the liver disease was associated with involvement of the alimentary tract (2 cases duodenal ulcer, 4 cases resected stomach). The presence of antibile autoantibodies implies the existence of a direct contact between the bile antigens and the immunoforming system, and therefore, their passage through the circulation. Two possible pathways for the penetration of the circulatory system by these antigens are proposed: 1) cholestasis; and 2) the antigens may be absorbed in the presence of proteolytic failure of the gastrointestinal tract.

0638 A LYMPHOCYTE-INHIBITING FACTOR ISOLATED FROM NORMAL HUMAN LIVER. *(E.)* Schumacher, K.; Maerker-Alzer, G.; Wehmer, U. (Med. Univ. Clin., Cologne, Germany). *Nature* 251(5476):655-656, 1974.

A leukocyte-inhibiting factor was isolated from the soluble supernatant after homogenization and centrifugation of normal human liver at 100,000 g for 90 min in Hanks solution, pH 7.2. A pure substance with inhibiting properties was obtained after elution and ion exchange chromatography. The inhibitor was a protein, molecular weight 65 x 10^3-8 x 10^3. Electrophoretic mobility was slow, similar to the β-gamma globulins. The protein was a potent inhibitor of lymphocyte transformation after stimulation by phytohemagglutin, purified protein derivative and by cellular antigens or transplantation antigens. It also inhibited the metabolism of *in vivo* activated lymphocytes and the migration of macrophages. After PHA stimulation, most of the *de novo* synthesis of RNA was inhibited. The specificity of lymphocyte metabolism inhibition seems to be very high: doses which caused a 100% inhibition of lymphocyte metabolism gave only a very slight reduction of the metabolic rate of HeLa cells. No species specificity of the inhibitor was found. The physiological significance of the inhibitor has not been determined.

0639 HL-A ANTIGEN DISTRIBUTION IN HEPATIC DISEASES. *(Ger.)* Freudenberg, K. J.; Erdmann, K.; Meyer zum Buschenfelde, K. H.; Forster, E.; Berger, J. (Med. Univ. Clin. Polyclin. II, Univ. Mainz, Germany). *Verh. Dtsch. Ges. Inn. Med.* 79: 645-646, 1973.

The distribution of the human leukocyte antigen (HL-A) was studied in patients with acute hepatitis, chronic persistent hepatitis, chronic aggressive hepatitis, and cirrhosis. Statistically significant increases were observed in the frequency of HL-A8 in patients with chronic aggressive hepatitis. The statistically insignificant increase in the frequency of HL-A12 determined in patients with chronic persistent hepatitis may be due to the small number of the patients tested. The findings indicate a coupling disequilibrium between the transplantation antigen HL-A8 and genetic factors that cause especially active immune reactions. It is concluded that carriers of this antigen are predisposed for autoimmune diseases.

0640 AUSTRALIA ANTIGEN, HL-A ANTIGENS, AND LYMPHOCYTE AUTOANTIBODIES IN CHRONIC AGGRESSIVE AND CHRONIC PERSISTENT HEPATITIS. *(Ger.)* Bertrams, J.; Reis, H. E.; Kuwert, E.; Selmair, H. (Dep. Med. Virol. Immunol., Univ. Essen, Germany). *Verh. Dtsch. Ges. Inn. Med.* 79:642-644, 1973.

The frequency of Australia antigen (HB_sAg), human leukocyte antigen (HL-A), and of cold reacting, complement-dependent, auto-lympho-cytotoxins was studied in 77 patients with chronic aggressive hepatitis and in 51 patients with chronic persistent hepatitis. The frequency of HL-A8, determined by Kissmeyer-Nielsen's microlymphocytotoxicity test, was 29.9% in group 1 patients and 21.6% in group 2 patients, *versus* a control value of 20.5%. The HL-A1, 8 frequency was 23.4% in the 1st group, 13.7% in group 2, and 15.8% for controls. HL-A-W-18 frequency was 25.5% in group 2 patients, 7.8% in group 1 patients, and 10% in controls. HB_sAg, determined by migration electrophoresis and micro-complement binding reaction, was found in 48% of group 1 patients, and in 72.6% of the patients in group 2. The frequency of auto-lympho-cytotoxins (CoCoCy), determined at 15 C, was 48.1% in the 1st group, 13.7% in the 2nd group, and 6% in controls. The auto-CoCoCy was found to have antibody and autoantibody properties, and was related to immunoglobulin M (IgM) without showing any correlation with the total serum IgM level. Absence of HL-A specificity, and an optimum reaction

temperature of 15 C were also determined. Significant correlation between the occurrence and concentration of HB_sAg and the course of chronic hepatitis was established. The presence of W-18 antigen in chronic persistent hepatitis may be related to the insufficient immune elimination of HB_sAg or of type MS-2 hepatitis virus. HL-A8 and the haplotype HL-A1,8 may be related to the autoimmune processes observed in chronic aggressive hepatitis. The findings suggest that the different manifestations of chronic aggressive and chronic persistent hepatitis are a result of the different, individual-specific immune response of the organism to infection with the pathogen of serum and infectious hepatitis, or to the autoantigens appearing during infection.

0641 CHRONIC LIVER DISEASE AND HEPATITIS-B ANTIGEN: A PROSPECTIVE STUDY. *(E.)* Van Waes, L.; Segers, J.; Van Egmond, J.; Van Nimmen, L.; Barbier, F.; Wieme, R.; Demeulenaere, L. (St. Andrew's Hosp., Tielt, Belgium). *Brit. Med. J.* 5298 (3):444-446, 1974.

Immunodiffusion or counterelectrophoresis assays for hepatitis B antigen (HB Ag) were performed on 86 patients (48 men and 38 women) with chronic liver disease during follow-up periods averaging 38 months to investigate differences in the clinical course of HB Ag-positive and HB Ag-negative patients. A diagnosis of cirrhosis was made in 41 of the patients (mean age 52.1 yr) and chronic hepatitis in 45 (mean age 34.8 yr). HB Ag was present in 19 of 45 patients (42.2%) with chronic hepatitis (5 out of 16 with chronic active hepatitis and 14 out of 29 with chronic persistent hepatitis) and in 20 of 41 cirrhotic patients upon admission. At the end of the study 13 of 36 patients still diagnosed as suffering from chronic hepatitis were HB Ag-positive. In 9 cases (6 chronic active and 3 chronic persistent) the chronic hepatitis had changed to cirrhosis, confirmed by peritoneoscopy and biopsy. Positive results for HB Ag were found in 18 out of 50 diagnosed cirrhotics at the end of the study. The clinical course and biochemical and histological findings in the positive and negative cases for the antigen were similar, suggesting that HB Ag-positive chronic liver disease is not a distinct clinical entity. No significant differences in the incidence of autoantibodies (antinuclear factor, smooth muscle antibody, and antimitochondrial antibody) were found between HB Ag-positive patients and those patients negative for the antigen. Fourteen of 34 patients with chronic hepatitis were positive for autoantibodies (7 also HB Ag-positive), while 16 of 35 patients with cirrhosis tested were positive (4 also HB Ag-positive). In the HB Ag-positive cases the antigen tended to persist for months and years. The finding of autoantibodies equally in positive and negative patients for HB Ag confirms previous observations that antigen and autoantibodies are not mutually exclusive. When no irreversible lesions exist, disappearance of the antigen may be a sign that liver disease will resolve.

0642 ANTIBODY PATTERN IN AUTOIMMUNE HEPATITIS: CLINICAL SIGNIFICANCE AND DIFFERENTIAL DIAGNOSTIC VALUE. *(Ger.)* Berg, P. A.; Marker, A.; Traunecker, U. (Tubingen Med. Clin., Germany). *Verh. Dtsch. Ges. Inn. Med.* 79:650-653, 1973.

The antibody pattern was studied in autoimmune hepatitis (primary biliary cirrhosis and chronic aggressive hepatitis) in 75 female and 5 male patients in an attempt to classify autoimmune hepatitis to a group with, and another group without mitochondrial antibodies. Mitochondrial antibodies were detected in 41 patients. Primary biliary cirrhosis was diagnosed in 24 cases, and chronic aggressive hepatitis or cryptogenic cirrhosis in 17 cases. Increased alkaline phosphatase levels were found in 38 patients. Antibodies against nuclei and/or smooth muscle were found to occur simultaneously in 10 patients with primary biliary cirrhosis, and in 13 patients with chronic aggressive hepatitis. Of the 39 patients in whom no mitochondrial antibodies were detected, 35 had chronic aggressive hepatitis, and prolonged increase of the alkaline phosphatase level was found in 2 cases. Blood typing in autoimmune hepatitis with known etiology revealed no significant differences in the distribution of groups A and O in HA Ag positive hepatitis, while predominance of group A was determined in primary biliary cirrhosis and in cases in which mitochondrial antibodies were detected. The frequency of group O was 17% in patients with primary biliary cirrhosis, and 50.9% in patients with "lupoid" hepatitis, *versus* the average 41-45%. Further studies will be necessary to determine whether the synchronous occurrence of antibodies against nuclei, smooth muscles, and mitochondria is an indication of a simultaneous process in liver parenchyma and in the bile ducts.

0643 PLASMA CORTICOSTEROID LEVELS IN CHRONIC HEPATITIS AND CIRRHOSIS. *(Rus.)* Sokolova, G. M.; Dobrova, A. S. (Cent. Inst. Postgrad. Med., Moscow, USSR). *Ter. Arkh.* 46(4):72-75, 1974.

The 17-, 21-oxy- and 20-ketosteroid levels in plasma and diurnal urine were measured in 41 male and 11 female patients with chronic inflammatory liver diseases (26 cases of postnecrotic cirrhosis, 13 cases of portal cirrhosis, 5 cases of biliary cirrhosis, and 7 cases of chronic hepatitis). Reduction of the oxyketosteroid level in the plasma to 8.2 µg%, and normal level in the urine with increased percentage of the free oxyketosteroid fraction were observed in postnecrotic cirrhosis in its subcompensated stage. The 17-ketosteroid level in the urine decreased to 7.4 mg/day. The plasma oxyketosteroid level was below normal in decompensated postnecrotic cirrhosis, but higher than in subcompensated cirrhosis. The total urinary excretion of oxyketosteroids was normal with increased percentage of free oxyketosteroids. Significant reduction of plasma oxyketosteroid levels, slight reduction of the urinary excretion of oxyketosteroids with increased percentage of free oxyketosteroids, and slightly increased 17-ketosteroid were observed in decompensated portal cirrhosis. Reduced plasma and urinary oxyketosteroid levels, and considerable reduction of 17-ketosteroid excretion were found in biliary cirrhosis. Negligible reduction of the plasma oxyketosteroid level, considerable de-

crease in the total oxyketosteroid excretion, and moderately increased excretion of free oxyketosteroids were established in chronic hepatitis. The reduced plasma oxycorticosteroid levels, observed in all patients, especially in those with chronic hepatitis, reflected the reduced function of the adrenal cortex. Increased free oxyketosteroid levels in the urine was a consequence of the disturbed inactivation and conjugation function of the liver, and further enhanced the inhibition of the adrenal cortex. No substantial difference was found between the plasma and urine oxyketosteroid levels in patients with splenomegaly and hypersplenism and in patients with splenomegaly alone.

0644 THE SPONTANEOUS LONG-TERM EVOLUTION OF CHRONIC AGGRESSIVE HEPATITIS. *(E.)* Knoblauch, M.; Ammann, R.; Clavadetscher, P.; Grob, P.; Schmid, M. (Univ. Hosp., Zurich, Switzerland). *Digestion* 10(4/5):360, 1974.

0645 GROSS IMPAIRMENT OF HEPATIC DRUG METABOLISM IN A PATIENT WITH CHRONIC LIVER DISEASE. *(E.)* Adjepon-Yamoah, K. K.; Nimmo, J.; Prescott, L. F. (R. Infirm., Edinburgh, Scotland). *Br. Med. J.* 4(5941):387-388, 1974.

0646 IMPAIRED T-LYMPHOCYTE FUNCTION IN CHRONIC HEPATITIS? *(E.)* Tolentino, P.; Pasino, M.; Braito, A.; Astaldi, Jr., A.; Giacchino, R. (Dep. Infect. Dis., Univ. Genoa, Italy). *Digestion* 10(4/5): 331, 1974.

0647 INFLUENCE OF RIFAMPICIN ON METABOLIC CLEARANCE OF HEXOBARBITAL AND TOLBUTAMIDE IN HEALTHY SUBJECTS AND IN PATIENTS WITH CHRONIC LIVER DISEASE. *(E.)* Zilly, W.; Breimer, D. D.; Keller, B.; Dosch, A.; Richter, E. (Dep. Intern. Med., Wurzburg Univ., Germany). *Digestion* 10(4/5):313, 1974.

0648 ANTIBODIES TO HUMAN PANCREAS IN LIVER DISEASES. *(E.)* Rizzetto, M.; Verme, G. (Mauriziano Hosp., Turin, Italy). *Digestion* 10(4/5):377, 1974.

See also, 0171, 0573, 0577, 0597, 0610, 0626, 0629, 0660.

LIVER AND BILIARY TRACT

Alcoholic Liver Disease

0649 STUDIES IN ALCOHOLIC LIVER DISEASE IN BRITAIN. I. CLINICAL AND PATHOLOGICAL PATTERNS RELATED TO NATURAL HISTORY. *(E.)* Brunt, P. W.; Kew, M. C.; Scheuer, P. J.; Sherlock, S. (R. Free Hosp. Med. Sch., London, England). *Gut* 15(1): 52-58, 1974.

A group of 258 British patients with various forms of alcoholic liver disease, such as steatosis, mild and severe hepatitis, and cirrhosis, was studied to investigate the clinical and histopathological forms of the disease, to relate these forms to the outcome, and to define factors which may influence prognosis. Of the 258 patients, 183 (71%) were males and 75 (29%) were females. There was no sex difference in the age distribution (mean of 50.0 yr and 50.4, resp.). Severity of the disease as judged histologically did not correlate well with clinical presentation although signs of hepatocellular failure were more common in severe hepatitis and cirrhosis. Fever, pigmentation, and clubbing were also indicators of these two conditions. It is suggested that alcoholic hepatitis is probably precirrhotic and carries a poor prognosis with laboratory signs of a moderate elevation of WBC and bilirubin. Poor survival in alcoholic liver disease was associated with continued drinking, the presence of active hepatitis, and probably with poor social (and nutritional) status.

0650 IMPAIRED ABSORPTION OF MICELLAR LONG-CHAIN FATTY ACID IN PATIENTS WITH ALCOHOLIC CIRRHOSIS. *(E.)* Malagelada, J. R.; Pihl, O.; Linscheer, W. G. (Tufts Univ. Sch. Med., Boston, Mass.). *Am. J. Dig. Dis.* 19(11):1016-1020, 1974.

Absorption of micellar long-chain fatty acid was diminshed in 12 chronic alcoholic patients with liver cirrhosis when compared with controls as demonstrated during perfusion of the proximal small bowel with a micellar solution of long-chain fatty acid (oleic). In the cirrhotic patients the rate of absorption of oleic acid was only 1.0 μM/min/cm compared with 1.8 μM/min/cm for the controls. Parallel experiments showed no difference in absorption of medium-chain fatty acids (octanoic): 5.7 μM/min/cm for cirrhotic patients and 7.1 μM/min/cm for controls. The results suggest impairment of the mucosal transport of long-chain fatty acid in cirrhosis. It is suggested that this abnormality, possibly related to portal or lymphatic hypertension, contributes to fat malabsorption and malnutrition in these patients.

0651 BILIARY DEOXYCHOLATE IN PATIENTS WITH ALCOHOLIC CIRRHOSIS. *(E.)* Mehta, S. J.; Struthers, Jr., J. E.; Kaye, M. D.; Naylor, J. L. (Denver VA Hosp., Colorado). *Gastroenterology* 67(4): 674-679, 1974.

Deoxycholate metabolism was studied in 10 patients with biopsy-proved alcoholic cirrhosis. Deoxycholate was absent from the bile of 3, present in reduced quantities in 3, and present in normal amounts in

4 patients as determined by ion-exchange column chromatography. The oral administration of cholic acid (1.0 g daily for 4 days) was accompanied by a rise in biliary deoxycholate from subnormal to normal levels in 3 patients but had little or no effect in the 3 whose bile initially contained no deoxycholate. The feeding of conjugated deoxycholate, 0.4 g daily for 4 days, to 2 of the latter patients resulted in the prompt appearance of deoxycholate in bile. As a result of these studies it was impossible to identify a single mechanism responsible for reduction of biliary deoxycholate in patients thus affected. Rather, it appears that both a relative lack of substrate (cholic acid) and deficient intestinal conversion of cholic acid to deoxycholate may contribute to the observed reductions in biliary deoxycholate.

0652 CLINICAL STUDIES OF ALCOHOLIC KETOACIDOSIS. *(E.)* Cooperman, M. T.; Davidoff, F.; Spark, R.; Pallotta, J. (Beth Israel Hosp., Boston, Mass.). *Diabetes* 23(5):433-439, 1974.

The clinical findings and biochemical changes in 6 nondiabetic female patients who had 7 episodes of severe ketoacidosis are reported. One pregnant patient had 2 episodes at 28 and 32 weeks gestation; all patients admitted to heavy chronic alcohol intake. On admission, the patients were conscious and alert. Mean values were 143 mg/100 ml for plasma glucose and 7.25 for arterial pH. Plasma bicarbonate was depressed with a mean anion gap of 18. ß-Hydroxybutyrate:acetoacetate ratio averaged 5.2. All patients had liver function abnormalities. Mean serum immunoreactive insulin was low, 5 µU/ml, cortisol was elevated at 76.5 µg%, and mean growth hormone level was 14.1 ng/ml. Free fatty acid concentration, measured on admission in 1 episode, was 1,945 mEq/liter. Therapy with glucose, saline, and minimal amounts of alkali led to prompt recovery. The preponderance of women reported with this condition suggests a possible role for ovarian and placental hormones in its pathogenesis. Fetal drain on carbohydrate reserves may further contribute to the tendency to ketosis.

0653 ASCORBIC-ACID SATURATION AND ETHANOL METABOLISM. *(E.)* Krasner, N.; Moore, M. R.; Dow, J.; Goldberg, A. (Stobhill Gen. Hosp., Glasgow, Scotland). *Lancet* 2(7882):693-694, 1974.

In a study of 12 patients with clinical or biochemical evidence of non-alcoholic liver disease, ascorbic-acid levels, measured as leukocyte ascorbic acid, were correlated with activities of hepatic alcohol dehydrogenase. The results showed a highly significant linear correlation between hepatic alcohol-dehydrogenase activity and leukocyte ascorbic acid levels with a correlation coefficient r = 0.88. In another series of experiments on healthy male volunteers, the clearance of ethanol from the blood (ß60) was measured before and after treatment with oral ascorbic acid in a dose of 1 g daily for 2 weeks. The results indicated that there was a significant rise in leukocyte ascorbic acid levels, except in 2 instances where they were depressed. There was a highly significant rise in ß60 except in those subjects with reduced levels of leukocyte ascorbic acid in whom the ß60 values were also depressed. A significant correlation was found between ß60 and leukocyte ascorbic acid levels r = 0.6. It seems therefore that there is an association between ascorbic acid and ethanol metabolism and that the enhanced rate of ethanol clearance from the blood may be associated with increased activities of alcohol dehydrogenase.

0654 LIVER STEATOSIS IN HUMAN PATHOLOGY. *(E.)* Popper, H. (Mt. Sinai Sch. Med., City Univ. New York, N.Y.). *Rend. Gastroenterol.* 5(3):213-214, 1973.

0655 FECAL CHYMOTRYPSIN IN ALCOHOLIC LIVER DISEASE. *(E.)* Moeller, D. D.; Dunn, G. D.; Klotz, A. P. (Univ. Kansas Med. Cent., Kansas City). *Am. J. Dig. Dis.* 19(12):1138-1142, 1974.

0656 EFFECT OF ETHANOL ON MITOCHONDRIAL ADENOSINE TRIPHOSPHATASE (ATPase) AND ALCOHOL DEHYDROGENASE (ADH) METABOLISM EVALUATED BY QUALITATIVE HISTOCHEMISTRY. *(E.)* Monteiro, E.; Cristina, M. L.; Batista, A.; Pinto Correia, J. (Santa Maria Hosp., Lisbon, Portugal). *Digestion* 10(4/5):369, 1974.

See also, 0130, 0134, 0256.

LIVER AND BILIARY TRACT

Cirrhosis

0657 HEPATIC CIRRHOSIS AND THE SUDDEN DEATH SYNDROME. *(E.)* Berkheiser, S. W. (Harrisburg Polyclin. Hosp., Pa.). *Aerosp. Med.* 45(8): 954-958, 1974.

An analysis of the 3,693 autopsies performed at a large general hospital between 1957 and 1967 disclosed 118 cases (3.2%) in which cirrhosis was clinically and pathologically significant. The average age of these 118 patients was 58 years. 74% were males. The cirrhosis was latent prior to death in 59%, and 32% died within 12 hours from the sudden onset of final symptoms. Typical portal cirrhosis was found in 81.4% of the patients, fatty cirrhosis in 11%, obstructive biliary cirrhosis in 5%, and hemochromatosis in 2.5%. Ulcerative colitis and cirrhosis were noted in only 3 patients (2.5%). Significant clinical findings included hepatomegaly (48%), splen-

omegaly (78%), and ascites (37%). Thirty-eight percent of the patients admitted to a history of chronic alcoholism. Among the 38 cases of sudden death, the primary immediate cause of death was cardiovascular disease in 39.5% of the patients, rupture of esophageal varices in 16%, liver failure in 13%, and gastrointestinal hemorrhage, pneumonia, and miscellaneous causes, 10.5% each. It is suggested that screening for unsuspected cirrhosis warrants serious consideration because of the rising incidence of this disease and its probability for producing sudden disability or death.

0658 PREGNANCY AND CIRRHOSIS OF THE LIVER. *(Ger.)* Manzl, J. (Reg. Hosp., Salzburg, Austria). *Zentralbl. Gynaekol.* 96(30):946-949, 1974.

After pregnancy and parturition without any complication, sudden death occurred in a para 5 due to intra-abdominal bleeding caused by a ruptured vein of the Plexus ovaricus sinister on the 4th postpartum day. Eutrophic, macronodular cirrhosis of the liver and tumor of the spleen were found on autopsy. Anastomoses between the origin of the portal vein and the veins of the pelvis results in the portal hypertension immediately involving the gonadal plexus in cirrhosis, thus causing rupture of the vein. Hypertension in the gonadal veins is further enhanced by varicose veins. The findings indicate the high probability of a causal relationship between varicose veins, cirrhosis, portal hypertension, and rupture of the gonadal vein.

0659 THE "CRYPTOGENETIC" FEVER OF ALCOHOLIC CIRRHOSIS. CLINICAL, LABORATORY AND PATHOLOGY CORRELATIONS IN 72 CASES. *(Fr.)* Bergerault, P.; Le Bodic, M. F.; Lenne, Y.; Le Bodic, L.; Oudea, P.; Miniconi, P. (St. Jacques Hosp., Nantes, France). *Ann. Gastroenterol. Hepatol. (Paris)* 9(6): 537-545, 1973.

Patients hospitalized for alcoholic cirrhosis were classified into 3 groups: 1) apyretic (91 cases, 27.3%); 2) with fever of known origin (171 cases, 51.2%); 3) with cryptogenetic fever (72 cases, 21.5%). All the patients underwent a thorough clinical examination and a number of laboratory and histologic tests. There were no significant clinical differences between the 3 groups. Of the laboratory test values, only the concentration of SGOT was significantly higher for group 3 than for the other 2 groups. Of the histologic tests, there were significantly higher levels of Mallory bodies in 60% of the cases of group 3 and cellular necrosis and infiltration of polynuclears in the parenchyme in 70% of the cases of group 3 compared with the other 2 groups. These histologic signs indicate subacute hepatitis, which has been suggested by other authors as a cause of cryptogenetic fever in alcoholic cirrhotics. To account for the 30% of the cases where these signs were not present, other authors have suggested subclinical intestinal infection, pyrogenic steroids, bacteria produced substances, and hepatic insufficiency.

0660 ANTI-T AGGLUTININ IN CIRRHOSIS OF THE LIVER. *(E.)* Boccardi, V.; Attina, D.; Bigliocchi, S.; Girelli, G. (Inst. Pathol., Med. I, Univ. Rome, Italy). *Vox Sang.* 26(1):83-85, 1974.

Titration for anti-T activity was performed on 46 patients of whom 44 had liver cirrhosis and 2 had advanced chronic active hepatitis. No significant change of titer was detected after storage of the samples for several months. In general, screening tests for irregular isoantibodies active on untreated and papain-treated red cells were negative. Compared with normal control sera, the anti-T titer in the cirrhotic sera was significantly elevated. All cirrhotic patients showed increased γ-globulin levels of the polyclonal peak type, but there was no correlation between γ-globulin level and anti-T titer. Similarly, the antibody titer was not related to clinical condition. In one chronic active hepatitis patient, the antibody titer fell from 8 to 2 after 5 months treatment with azathioprine; a control biopsy revealed histological improvement with a fall in γ-globulins. Anti-T also fell from 8 to 1 in a patient treated 3 months with β-metasone, but there was no significant alteration in the γ-globulin level. There were no changes in titer in 9 other patients undergoing no immunosuppressive treatment.

0661 MORPHOLOGICAL CHANGES IN SMALL INTESTINAL MUCOSA IN CIRRHOSIS. *(Sp.)* Herrerias Gutierrez, J. M.; Jimenez Pereperez, J.; Damiano Rivero, A.; Jurado Nieto-Guerrero, M. (Fac. Med., Cadiz, Spain). *Rev. Esp. Enferm. Apar. Dig.* 43(6): 603-610, 1974.

The structure of jejunal mucosa was studied in biopsies taken from 20 patients (16 males and 4 females, aged 16-72 yr) with histologically confirmed septal, micronodular cirrhosis and diarrhea. Peroral aspiration biopsies were taken with a modified Crosby-Kugler capsule distal to the angle of Treitz 60 min after administration of metoclopramide (10 mg i.v.). The pattern of the jejunal mucosa was normal in 40% of the biopsies. Abnormalities consisted of epithelial stasis and edema (10%), villous stasis (10%), and epithelial edema with changes in the villi (10%). Findings typical of sprue were present in 10%, inflammation in 10%, sclerosis of the lamina propria in 5%, and epithelial ulcerations with flattening of the villi in 5%.

0662 PERITONEO-VENOUS SHUNTING FOR ASCITES. *(E.)* LeVeen, H. H.; Christoudias, G.; Ip, M.; Luft, R.; Falk, G.; Grosberg, S. (VA Hosp., Brooklyn, N.Y.). *Ann. Surg.* 180(4):580-591, 1974.

A new minor surgical procedure for ascites is described wherein a 1-way pressure activated valve is implanted to create a permanent peritoneo-venous shunt. The normally closed valve opens only when the peritoneal pressure rises 3-5 cm higher than the intrathoracic venous pressure, thus preventing backflow of blood and closing of the valve should the venous pressure rise from the overinfusion of ascitic fluid. The patient is started on respiratory ex-

ercises (reclining position) 24-48 hr after the operation which consist of inspiring through a tube against a resistance of 5 cm of water. The exercise is continued postoperatively for 15 min, 4 times a day, to take advantage of the pumping action created by the negative intrathoracic pressure. Cooperation of the patient during the exercises is essential in eliminating all of the ascites. Excluding immediate deaths from end stage disease (9 patients) and infections necessitating valve removal (2 patients), the procedure was successful in 26 out of a remaining 34 cases. One-third of the shunts became occluded usually 1-2 weeks postoperatively after the ascites had completely disappeared. Leakage of ascitic fluid around the threads of the valve allowing blood to replace the ascitic fluid in the venous tubing accounted for 90% of the failures. This defect has been corrected by sealing the threads. Occlusion of the shunt produces only temporary failure and requires prompt valve replacement.

0663 ORIGIN OF FAT IN CHYLOUS ASCITES OF PATIENTS WITH LIVER CIRRHOSIS. *(E.)* Malagelada, J. R.; Iber, F. L.; Linscheer, W. G. (Lemuel Shattuck Hosp., Tufts Univ. Sch. Med., Boston, Mass.). *Gastroenterology* 67(5):878-886, 1974.

The origin of fat in chylous ascites of cirrhotic patients was investigated by feeding medium and long chain fatty acids (20 mM Trioctanoin labeled with 50 μCi ^{14}C-octanoic acid and 20 mM Triolein labeled with 50 μCi ^{3}H-oleic acid). The patients included 3 cirrhotic patients with chylous ascites (2 men and 1 woman, aged 42-70 yr), 8 patients with cirrhosis and non-chylous ascites (7 men and 1 woman, aged 42-66 yr), and 4 noncirrhotic patients with chylous ascites (2 women and 2 men, average age 49 yr). The time of appearance (1 hr after feeding), time interval from ingestion to peak levels (5 hr), and half-life time of decay (58 hr) of labeled long chain fatty acids in ascitic chylomicrons were similar in all groups of patients. In the cirrhotic patients, however, the peak activity of long chain fatty acids found in chylous ascites (8% of ingested radioactivity) was much greater than in clear ascites (0.6%). An even greater amount of ingested long chain fatty acids was found in the chylous ascites of noncirrhotic patients (22%). More than 80% of the ingested labeled oleic acid present in chylous ascites was incorporated in esterified form in chylomicrons as compared to less than 50% in clear ascitic fluid. This significantly higher incorporation of ^{3}H radioactivity in subjects with chylous ascites when serum levels were identical suggests that intestinal lymph was the main source of ascitic chylomicrons. The appearance, concentration, and decay rate in ascites of ^{14}C-octanoic acid not incorporated in the chylomicrons was similar in cirrhotic patients with clear or chylous ascitic fluid, suggesting a similar pathway for transport of the non-protein-bound labeled octanoic acid. Within 5 hr after administration, free octanoic acid reached peak concentrations in both groups, followed by a rapid decline with only very small amounts (less than 25% of peak remaining after 12 hr). A relatively low proportion of triglycerides were found in clear ascitic fluid (37 *versus* 295 for chylous cirrhotics and 898 for chylous noncirrhotics), indicating that most of the triglycerides were present in the smaller particles, probably a combination of small chylomicrons and very low, low, and high density lipoproteins. Only small chylomicrons, probably leaking from the intestinal lymphatics, appeared to enter the ascites of cirrhotic patients with clear ascitic fluid. When the number and size of these chylomicrons increases, the ascitic fluid acquires a chylous nature. Whether the underlying pathogenesis involves abnormal permeability or rupture of the lymphatics is unknown, but the process may reverse with improvement of liver function.

0664 HEMORRHAGE FROM A PORTAL-SYSTEMIC VENOUS SHUNT WITH UNUSUAL LOCALIZATION IN A CASE OF PORTAL HYPERTENSION. *(E.)* Borjesson, B.; Olsson, A. M.; Vang, J. O. (Univ. Hosp., Lund, Sweden). *Scand. J. Gastroenterol.* 9(6):571-573, 1974.

Hemorrhage from a portal-systemic collateral vein leading from the mesentery of the ileum to the lateral right pelvic wall is described for a 48-yr-old woman with portal hypertension. The patient had previously undergone a subtotal gastrectomy *ad modum* Billroth II for accidental hydrochloric acid ingestion (27 yr prior), a vagotomy after development of an ulcer (16 yr prior), and a Billroth I reconstruction following diagnosis of a new stomal ulcer and a stomal stricture (9 yr prior). The patient had also been previously admitted (4 yr prior) because of hematemesis and melena. Surgical exploration at this prior admission had revealed a cirrhotic liver which was cryptogenic, and splenoportography had shown varicose veins in the esophagus. The patient was readmitted 4 yr after these observations because of melena recurrence, followed by the development of hepatic coma a few days later. Vasopressin infusion in 2 instances and Sengstaken tube treatment in 1 instance were successful in stopping the bleeding for periods of about 2 days. Angiography of the celiac axis and the superior and inferior mesenteric arteries revealed varicose veins around the duodenum and terminal part of the ileum. During abdominal exploration following onset of the symptoms of ileus, a sigmoid volvulus was found along with adhesion of the last part of the ileum to the region of the right ovary. During dissection, a finger-wide vein was found leading from the mesentery of the ileum, *via* the ileum wall, to the lateral right pelvic wall. Opening of the ileum during resection revealed the vein bulging into the lumen, with a blood clot appearing in a hole located in the vein wall. The resection was uneventful, and the patient has had no further hemorrhages (18 months after operation). The bleeding portal-systemic collateral probably developed in a postoperative adhesion. The difficulty in evaluating a given treatment for such patients is illustrated by the fact that on 1 occasion a Sengstaken tube promptly stopped the bleeding even though there was not even a theoretical possibility of its influencing the hemorrhage.

0665 CLINICO-MORPHOLOGICAL CORRELATIONS IN THE PORTAL HYPERTENSION SYNDROME DUE TO SUBHEPATIC OBSTRUCTION. *(Rus.)* Simonov, V. V.; Kalashnikov, S. A. (S.M. Kirov Mil. Med. Acad., Leningrad, USSR). *Vestn. Khir.* 112(1):41-44, 1974.

Morphological liver studies of 44 patients with subhepatic obstruction of the portal blood flow revealed one case of cirrhosis. Vena portal thrombosis was the main cause of the subhepatic obstruction (15 cases). Findings show that hypertension caused by subhepatic obstruction did not result in cirrhosis. Marginal biopsy in the 15 cases did not indicate any morphological changes of the liver. Hepatic changes in the remaining cases were intumescence of periportal interlayer outgrowths, and evidence of lymphoidohistiocytary infiltrations which were observed in other lesions. In some cases hepatic cell dystrophy may be related to the impaired portal blood flow. No relation was found between the cause of the subhepatic obstruction and the characteristic clinical symptoms.

0666 PROGRESS REPORT: MITOCHONDRIAL ANTIBODIES (AMA). *(E.)* Doniach, D.; Walker, G. (Middlesex Hosp., London, England). *Gut* 15(8):664-668, 1974.

The use of mitochondrial antibodies as a diagnostic tool for the detection of primary biliary cirrhosis is reviewed. These antibodies have been detected in 84-96% of patients with primary biliary cirrhosis. They occur in patients with cryptogenic cirrhosis, in some patients with active chronic hepatitis, in a small percentage of patients with various collagenoses, and in the idiopathic autoimmune disorders of the thyroiditis/gastritis/adrenalitis group. Two systemic lupus erythematosus-like syndromes without liver involvement have been identified in which mitochondrial antibodies are a feature. Aside from these disorders, several large epidemiological studies have confirmed the rarity of mitochondrial antibodies in the general population and also in patients with cirrhosis of types other than those discussed above. An explanation for the reported positive mitochondrial antibodies in extrahepatic biliary disease may be found in the number of immunofluorescence patterns which may be produced in the tissues of different species with human sera; for instance, at least 6 discrete reactions may resemble those produced by mitochondrial antibodies on rat kidney and stomach sections, which are frequently used as substrates for mitochondrial antibody tests. In the context of sera from liver patients, the most important reaction to be differentiated is that produced by the microsomal antibodies. Other patterns resembling those due to mitochondrial antibodies are unconnected with liver disease. The origin of mitochondrial antibodies is still largely unexplained but it is possible that a virus or related particle plays a causal role in autoimmune disorders or that some phage-like organism invades the mitochondria in bile duct cells in primary biliary disease.

0667 THE EPIDEMIOLOGY OF PRIMARY BILIARY CIRRHOSIS: A SURVEY OF MORTALITY IN ENGLAND AND WALES. *(E.)* Hamlyn, A. N.; Sherlock, S. (Roy. Free Hosp., London, England). *Gut* 15(6):473-479, 1974.

Over a 5-yr period (1967-1971) deaths from primary biliary cirrhosis in England and Wales amounted to 165 (146 women and 19 men). This corresponds to a crude death rate of 1.2/million/yr for females or 0.6/million/yr for both sexes. Deaths rose sharply at ages 50-54, where mortality was 4.1/million/yr, with a secondary peak to ages 70-74. Standardized mortality ratios varied from 52 (i.e., 52% of the national average) in the North of England to 141 in the London area. No relation existed between regional mortality and altitude, rainfall, annual temperature variation, soil type, or the presence of heavy industry or mining. There was a suggestive excess of deaths (134) among married women. The greater frequency of deaths in the London area, a rise in mortality from country to urban areas, a fall-off in deaths from primary biliary cirrhosis in old age, and predominance for social class I (professional occupations) suggest a simple relation with standards of medical care or diagnosis. An 'epidemic' of deaths in 1971 is attributed to greater availability of the mitochondria antibody test in the regions. Subjective impressions that primary biliary cirrhosis was more prevalent in Wales than in England were not confirmed. If genetic mechanisms are important, as appears from familial reports, they must be widely distributed in the general population.

0668 USE OF BUMETANIDE IN THE TREATMENT OF ACITES [SIC!] DUE TO LIVER DISEASE. *(E.)* Moult, P. J. A.; Lunzer, M. R.; Trash, D. B.; Sherlock, S. (R. Free Hosp., London, England). *Digestion* 10(4/5):370, 1974.

0669 ADHERENCE OF COAGULATION FACTORS TO THE DIALYSIS MEMBRANE OF THE ASCITES REINFUSION MACHINE: EXPLANATION FOR PROLONGATION OF PROTHROMBIN TIME. *(E.)* Wilkinson, S. P.; Portmann, B.; Henderson, J.; Williams, R. (King's Coll. Hosp., London, England). *Digestion* 10(4/5):387, 1974.

0670 CYSTIC FIBROSIS MANIFESTED AS FOCAL BILIARY CIRRHOSIS IN A NEWBORN INFANT WITH CONGENITAL HEART DISEASE. *(E.)* Cooper, H. S.; Oppenheimer, E. H. (John Hopkins Univ. Sch. Med., Hosp., Baltimore, Md.). *John Hopkins Med. J.* 135(4):268-273, 1974.

0671 THE BILIARY SYSTEM IN PRIMARY BILIARY CIRRHOSIS: A STUDY BY ENDOSCOPIC RETROGRADE CHOLANGIOPANCREATOGRAPHY (ERCP). *(E.)* Summerfield, J. A.; Elias, E.; Dick, R.; Sherlock, S. (R. Free Hosp., London, England). *Digestion* 10(4/5):381-382, 1974.

0672 ANATOMIC BASIS OF THE SURGICAL TREATMENT OF PORTAL HYPERTENSION. *(E.)* Salam, A. A.; Warren, W. D. (Emory Univ. Sch. Med., Atlanta, Ga.). *Surg. Clin. North Am.* 54(6):1247-1257, 1974.

0673 SPLENIC VEIN THROMBOSIS, SEGMENTAL PORTAL HYPERTENSION AND BLEEDING ESOPHAGEAL VARICES PRODUCED BY A CONGENITAL PANCREATIC CYST. *(E.)* Wolloch, Y.; Chaimoff, C.; Lubin, E.; Dintsman, M. (Beilinson Hosp., Petah Tikva, Israel). *Isr. J. Med. Sci.* 10(6):670-673, 1974.

0674 PORTAL HYPERTENSION WITH VARICES IN ECTOPIC SITES. *(E.)* Hamlyn, A. N.; Lunzer, M. R.; Morris, J. S.; Puritz, H.; Dick, R. (R. Free Hosp., London, England). *Digestion* 10(4/5):353-354, 1974.

0675 MORPHOLOGICAL FEATURES OF INDIAN CHILDHOOD CIRRHOSIS: THE SPECTRUM OF CHANGES AND THEIR SIGNIFICANCE. *(E.)* Aikat, B. K.; Bhattacharya, T.; Walia, B. N. S. (Postgrad. Inst. Med. Educ. Res., Chandigarh, India). *Indian J. Med. Res.* 62(7):953-963, 1974.

0676 KINETIC STUDIES OF THE METABOLISM OF UNCONJUGATED BILIRUBIN IN PATIENTS WITH COMPENSATED CIRRHOSIS. *(E.)* Owens, D.; Jones, E. A.; Richards, T. G.; Carson, E. (Dep. Med., Univ. Liverpool, England). *Digestion* 10(4/5):372-373, 1974.

0677 THE URINE EXCRETION OF HYDROXYPROLINE BY CIRRHOSIS OF THE LIVER. *(E.)* Dinkov, I.; Conev, K.; Mineev, M. (I.P. Pavlov Higher Med. Inst., Plovdiv, Bulgaria). *Folia Med. (Plovdiv)* 15(3):151-155, 1973.

0678 SERUM COPPER IN COMPLEX INVESTIGATIONS OF LIVER CIRRHOSIS. *(E.)* Mincev, M.; Geneva, B.; Kostova, S. (I.P. Pavlov Higher Med. Inst., Plovdiv, Bulgaria). *Folia Med. (Plovdiv)* 15(5): 265-269, 1973.

0679 ABNORMALITY IN TISSUE FERRITIN IN IDIOPATHIC HAEMOCHROMATOSIS. *(E.)* Powell, L. W.; Alpert, E.; Drysdale, J. W.; Isselbacher, K. J. (Massachusetts Gen. Hosp., Boston). *Aust. NZ J. Med.* 4(3):303-304, 1974.

0680 THE ABNORMAL LEVEL OF ISOENZYMES OF PLASMA MONOAMINE OXIDASE (MAO) IN FIBROTIC LIVER DISEASE. *(E.)* Lin, A. M.; Davis, L. M.; Castell, D. O. (U.S. Nav. Hosp., Philadelphia, Pa.). *Gastroenterology* 67(4):810, 1974.

0681 LIVER CIRRHOSIS. *(Fl.)* De Groote, J. (St. Rafael Gen. Hosp., Louvain, Belgium). *Tijdschr. Geneesk.* 29(14):687-690, 1973.

0682 MESOCAVAL AND PORTACAVAL SHUNTS USING PANELLED BOVINE ARTERIAL H GRAFTS. *(E.)* Rosenberg, N.; Konigsberg, S. F.; Noronha, J. (Rutgers Med. Sch., New Brunswick, N.J.). *Arch. Surg.* 109(6): 754-758, 1974.

0683 FAMILIAL OCCURRENCE OF A MEMBRANE IN THE SUPRAHEPATIC SEGMENT OF THE INFERIOR VENA CAVA (IVC). *(E.)* Erkelens, D. W.; Homan van der Heide, J. N.; Potocky, V.; Beekhuis, G.; Gips, C. H. (Univ. Hosp., Groningen, Netherlands). *Digestion* 10(4/5):347-348, 1974.

0684 BIOCOMPATIBILITY OF RESIN PERFUSION FOR THE REMOVAL OF CHOLEPHILIC ANIONS FROM THE PLASMA IN LIVER FAILURE: EXPERIMENTAL AND CLINICAL STUDIES. *(E.)* Weston, M. J.; Mellon, P.; Langley, P. G.; Williams, R. (King's Coll. Hosp., London, England). *Digestion* 10(4/5):386, 1974.

0685 WHAT IS THE REASON FOR THE CHARACTERISTIC SCAN ABNORMALITY SEEN WITH CHRONIC LIVER DISEASE? *(E.)* Horisawa, M.; Goldstein, G.; Waxman, A.; Reynolds, T. (Univ. South. California Sch. Med., Los Angeles). *Gastroenterology* 67(4): 799, 1974.

See also, 0153, 0195, 0196, 0380, 0381, 0382, 0383, 0444, 0569, 0572, 0573, 0577, 0584, 0590, 0635, 0641, 0643, 0648.

LIVER AND BILIARY TRACT

Gallbladder and Biliary Tract Diseases

0686 FACTORS INFLUENCING THE LONG TERM RESULTS OF CHOLECYSTECTOMY. *(E.)* Stefanini, P.; Carboni, M.; Patrassi, N.; Loriga, P.; De Bernardinis, G.; Negro, P. (Univ. Rome Med. Sch., Italy). *Surg. Gynecol. Obstet.* 139(5):734-738, 1974.

Eight hundred patients who underwent cholecystectomy during the period 1960 to 1973 were evaluated in a follow-up study to establish the frequency and severity of postcholecystectomy symptoms and to analyze the possible factors influencing these distresses. The longest follow-up period was 12 yr 6 months, the average time being 4 yr 2 months. There were 551 patients who had symptomatic cure; 217, mild symptoms; and 32, severe symptoms. Residual stones in the common bile duct and stenosis at the sphincter of Oddi were found to be the underlying causes of severe postcholecystectomy distress. Telecholangioscopy associated with cholangiography was helpful in detecting common bile duct diseases. The routine use of this procedure decreased overlooked biliary disorders from 8 to 2%. The procedure of choice in the treatment of residual stones in the common bile duct and stenosis at the sphincter of Oddi was

sphincteroplasty, which provided excellent long term results with a low operative motality rate. Mild postcholecystectomy symptoms were found to be related to hepatic damage which usually is present in patients with gallbladder disease. The frequency of these distresses was significantly influenced by the degree of hepatic damage present before operation. Patients with a long interval between onset of symptoms and cholecystectomy and with severe inflammation of the gallbladder had a higher incidence of mild distress.

0687 DYSFUNCTION OF THE SPHINCTER OF ODDI IN A POSTCHOLECYSTECTOMY PATIENT. *(E.)* Manier, J. W.; Cohen, W. N.; Printen, K. J. (Univ. Iowa Hosp. Clin., Iowa City). *Am. J. Gastroenterol.* 62(2):148-150, 1974.

A postcholecystectomy patient with recurrent episodes of right upper quadrant pain associated with transient elevations of the SGOT, LDH and alkaline phosphatase was diagnosed as having a motor malfunction of his sphincter of Oddi. During an i.v. cholangiogram, he developed radiographically demonstrated ductal distention and pain. A subsequent exploration of his duct was negative. An operative cholangiogram revealed a mildly dilated duct which emptied slowly into the duodenum. A choledochoduodenostomy was performed. The postoperative course was uncomplicated and at a 4-month follow-up, the patient was well without recurrence of his original symptoms.

0688 CHOLESTASIS ASSOCIATED WITH EXTRABILIARY HODGKIN'S DISEASE: REPORT OF THREE CASES AND REVIEW OF FOUR OTHERS. *(E.)* Perera, D. R.; Greene, M. L.; Fenster, L. F. (Dep. Med., Univ. Washington, Seattle). *Gastroenterology* 67(4):680-685, 1974.

Case histories are reported for 3 patients (all women ranging in age from 21-36 yr) with Hodgkin's disease who developed idiopathic cholestasis, and the salient features of 4 cases previously described in the literature are reviewed. Although idiopathic cholestasis is usually associated with advanced lymphomas, 2/3 patients had cholestatic jaundice during early Hodgkin's disease (stages I and IIA), and their jaundice improved during localized radiotherapy. All 7 patients had at least 1 laparotomy; all had liver biopsies, and 6/7 had operative cholangiograms. Evidence for Hodgkin's disease was not present on liver biopsy or at gross inspection of the liver at surgery, and the biliary tract was patent in every case. All liver biopsies demonstrated cholestasis. Five patients died, the time from onset of jaundice to death being 5-19 months. At postmortem examination, stage III or IV Hodgkin's disease was present in all 5 patients. In no instance was the biliary tract compromised or the liver extensively infiltrated by tumor. It is suggested that Hodgkin's disease be added to the differential diagnosis of patients with unexplained cholestatic jaundice.

0689 MEDICAL THERAPY FOR GALLSTONES. *(E.)* Schoenfield, L. J. (Cedars-Sinai Med. Cent., Los Angeles, Calif.). *Gastroenterology* 67(4):725-729, 1974.

The current status of research efforts directed towards elucidating the pathogenesis of cholesterol gallstones, evaluating agents, especially chenodeoxycholic acids (CDC) for their dissolution, and defining high risk groups that are appropriate for prophylaxis are reviewed as well as the potential for the medical treatment of gallstones. The pathogenesis of pigment gallstones which account for 10% of all stones analyzed in U.S. differs from that of cholesterol gallstones which comprise 90% of U. S. stones (80% mixed cholesterol stones and 10% pure cholesterol). The bulk of the report, therefore, deals with cholesterol gallstones. In the first trial of CDC as a treatment for cholesterol cholelithiasis, the dose of CDC ranged from 1.5 to 4.5 g/day. Later trials used 750 mg/day but the lowest therapeutic dose has not yet been determined. Biliary bile acid and lipid composition return to pretreatment levels within 1 month of discontinuing therapy; gallstones recurred in 2/5 patients off therapy for 1 yr. After dissolution, therefore, continuous or intermittent prophylactic therapy is necessary. The mechanism whereby CDC induces desaturation of bile, and thereby gallstone dissolution, is still undergoing investigation. CDC may induce liver damage, diarrhea, and ductal obstruction. Dissolution treatment of gallstones with CDC is still experimental. Efficacy is virtually certain but safety remains to be shown in a large scale, cooperative therapeutic trial. Such a trial is scheduled to begin in January, 1975 for a 2-yr period involving 900 patients who will receive 1 of 2 doses of CDC or placebo at 10 U.S. centers.

0690 ENDOSCOPIC PAPILLOTOMY AND REMOVAL OF GALLSTONES: ANIMAL EXPERIMENTS AND PRELIMINARY CLINICAL RESULTS. *(Ger.)* Demling, L.; Koch, H.; Classen, M.; Belohlavek, D.; Schaffner, O.; Schwamberger, K.; Stolte, M. (Med. Clin., Univ. Erlangen-Nuremberg, Erlangen, Germany). *Dtsch. Med. Wochenschr.* 99(45):2255-2257, 1974.

Endoscopic papillotomy and gallstone removal were performed in dogs and in 17 patients, including 3 cases of stenosis of the papilla of Vater, and 14 cases of cholelithiasis in the common bile duct. Transduodenal papillotomy was performed with a high-frequency diathermy catheter similar to that used for retrograde cholangiopancreatography, using a steel wire loop. The stones passed from the common bile duct spontaneously after 3 days, and they were easily extracted by means of Dormia's basket in another 5 cases. Endoscopic papillotomy, though primarily intended for elderly patients, is also indicated in younger patients, especially for repeated intervention after conventional operation.

0691 MULTIPLE BILIARY PAPILLOMATOSIS. *(E.)* Madden, Jr., J. J.; Smith, G. W. (Univ. Virginia Sch. Med., Charlottesville). *Cancer* 34(4):1316-1320, 1974.

A case of multiple biliary papillomata with nuclear atypical changes involving the entire biliary duct system is presented. The patient, a 50-yr-old male, had undergone cholecystectomy for a nonfunctioning gallbladder 2 yr prior and had suffered from acute pancreatitis 3 months prior, followed by the development of obstructive jaundice. Upon exploration of the common bile duct, a spongy papillary tumor was removed. The patient subsequently underwent a left hepatic lobectomy for a tumor in the left hepatic duct system detected by a T-tube cholangiogram. Subsequent transhepatic cholangiography revealed a large recurrent tumor mass almost entirely obstructing the common bile duct at its midportion. A smaller tumor mass distally in the common bile duct was also observed along with obstruction secondary to papillomata in at least 1 duct of the right liver lobe and 1 duct of the caudate lobe. A large quantity of papillomatous material was removed from the common and intrahepatic bile ducts during an exploratory laparotomy, and a choledochoduodenostomy was then performed to allow for more adequate drainage. The patient subsequently had several attacks of cholangitis with obstruction secondary to tumor involvement of the common bile duct, as demonstrated on an upper gastrointestinal series, and was again relieved by curettage. Weekly treatments with 98 or 100 mg of 1,3-bis(2-choroethyl)-1-nitrosourea were successful in decreasing the tumor mass, but intermittent jaundice and cholangitis persisted. The patient died with *Bacteroides* septicemia and shock 5 yr after the initial diagnosis.

0692 BILIARY INFECTIONS AND THE CHOICE OF ANTIBIOTICS. *(E.)* Ram, M. D.; Gharavi, M. A. (Huron Road Hosp., Cleveland, Ohio). *Am. J. Gastroenterol.* 62(2):134-138, 1974.

In a prospective study of 200 patients (66 men and 134 women, ranging in age from 19-90 yr) undergoing cholecystectomy, gallbladder bile was obtained at surgery under sterile conditions and was plated immediately for culture. Of the 200 patients, 189 were operated on for cholecystitis (acute and/or chronic) and the remaining 11 for other lesions. In the whole group, 32% of the specimens yielded a positive growth; in those with acute cholecystitis 65% had a positive culture. The proportion of positive growths increased with age. Among patients with cholecystitis and gallstones only 27% had a positive bacterial growth, while in those with cholecystitis not associated with gallstones, 43% had a positive bacterial growth. *E. coli* was the most frequent organism isolated. Sensitivity of the organisms to a variety of antibiotics was studied by the disc sensitivity method and zones of inhibition were measured and compared to standards. It was found that kanamycin, gentamicin and colistin were effective against all organisms isolated.

0693 THE NATURE OF THE SERUM ALKALINE PHOSPHATASES IN LIVER DISEASES. *(E.)* Price, C. P.; Sammons, H. G. (East Birmingham Hosp., England). *J. Clin. Pathol.* 27(5):392-398, 1974.

Sera were obtained from 58 patients with extrahepatic biliary obstruction and from 20 control subjects; homogenized liver and intestine samples served as tissue controls. Hepatic bile was obtained from 12 patients after cholecystectomy. The main serum alkaline phosphatase isoenzyme migrated in polyacrylamide gel to a position equivalent to the liver tissue isoenzyme and in front of the bone isoenzyme. Another isoenzyme was found at the origin, contributing at least 20% to the total activity. Three additional isoenzyme bands were detected in much smaller quantities. The main serum isoenzyme on cellulose acetate electrophoresis was in the α_2-globulin region with a less prominent band in the α_1-globulin area; the latter fraction was not found in normal control serum. Gel filtration studies of patient sera showed 2 peaks of alkaline phosphatase activity equivalent to the 19S and 7S protein fractions. In the 8 samples studied there was a good correlation between the contribution of the 'origin' isoenzymes on electrophoresis and the 19S fraction, which ranged between 12 and 42% of the total activity. In samples of patient sera, ultracentrifugation gave 2 fractions of alkaline phosphatase activity, one in the 7S protein fraction and one floating at the top of the sucrose gradient. The floating fraction after ultracentrifugation on electrophoresis was found to correspond with the 'origin' isoenzymes previously observed. These results indicate that there is an abnormal isoenzyme in the serum from patients with extrahepatic biliary obstruction and that this isoenzyme is similar but not identical with the main bile enzyme. It is suggested that it may be associated with a lipoprotein carrier.

0694 EFFECT OF CHOLIC ACID FEEDING ON BILE ACID KINETICS AND NEUTRAL FECAL STEROID EXCRETION IN HYPERLIPOPROTEINEMIA (TYPES II AND IV). *(E.)* Einarsson, K.; Hellstrom, K.; Kallner, M. (Dep. Med., Serafim Hosp., Stockholm, Sweden). *Metabolism* 23 (9):863-874, 1974.

Bile acid kinetics and the fecal excretion of neutral steroids were characterized in 10 hyperlipemic patients (6 men and 4 women, ages 45-65 yr) before and after p.o. administration of 0.8-1.0 g cholic acid/day for 2-3 weeks. Prior to cholic acid feeding, the turnover of cholic acid in 5 patients (4 women and 1 man) with type II hyperlipoproteinemia (178 mg/day) was significantly less than that for 5 men with type IV lipoprotein patterns (700 mg/day). The 2 groups did not differ with regard to pool size and half-life of cholic acid or pool size, half-life, and turnover of chenodeoxycholic acid. Cholic acid administration to type II patients resulted in markedly elevated cholic acid pool size (from 818-3230 mg), though cholic acid half-life was unaffected. This change in pool size was associated with a decreased pool size and turnover of chenodeoxycholic acid (from 892-465 and from 170-93, resp.), a reduced serum cholesterol level (from 348-310 mg/100 ml), reduced serum triglycerides (from 291-253 mg/100 ml), and an increased excretion of neutral steroids in the feces (from 327-557 mg/day). In contrast only 3 of the subjects with type IV lipoprotein patterns displayed an increase cholic acid

pool size, with the overall mean increase for the group being from 1174-1609 mg. Chenodeoxycholic acid turnover for the group fell from 278-163 mg/day. The fecal excretion of neutral steroids increased in all 5 (from 469-807 mg/day). Patients with types II and IV hyperlipoproteinemia appear to differ in their capacity to retain cholic acid, with type II patients having the ability to control chenodeoxycholic acid synthesis by feedback inhibition during p.o. administration of cholic acid. This latter ability may also be the case for type IV patients, though it was not significantly demonstrated in the present experiments.

0695 CHARACTERISTICS OF THE SHAPE OF THE PORTA HEPATIS IN RELATION TO THE SIZE OF THE LIVER. *(Ger.)* Bauer, J. A.; Hellerer, O. (Anat. Inst., Univ. Munich, Germany). *Munch. Med. Wochenschr.* 116(43):1881-1884, 1974.

The characteristics of the shape of the porta hepatis in relation to the size of the liver, the mode of junction of the hepatic duct, and the mode of division of the portal vein were investigated in 84 visceral sites in adults with and without hepatomegaly. The junction of the right and left hepatic ducts with the common hepatic duct corresponded to the size of the liver in 61 cases (73%), i.e., extrahepatic localization without hepatomegaly was found in 28 cases, and intrahepatic localization was found in 33 cases in hepatomegaly. The cystic duct joined with the common hepatic duct in 74 cases, and with the right hepatic duct in 6 cases. The branching of the portal vein was extrahepatic in 32 cases without hepatomegaly, and intrahepatic in 23 cases in hepatomegaly. The branching of the hepatic artery was extrahepatic in 46 cases without hepatomegaly, and intrahepatic in 7 cases in hepatomegalia. The extrahepatic junction of the right and left hepatic ducts with the common hepatic duct was always Y-shaped, while both Y-shaped and T-shaped junctions occurred in intrahepatic junction. The branching of both the portal vein and hepatic artery was Y-shaped both with and without hepatomegaly in the case of extrahepatic localization. The intrahepatic branching of the hepatic artery was Y-shaped both with and without hepatomegaly, while the intrahepatic branching of the portal vein was Y-shaped in 22 cases without hepatomegaly, in 15 cases with hepatomegaly, and T-shaped in 2 cases without hepatomegaly, and in 8 cases with hepatomegaly. The findings indicate that extrahepatic junctions and divisions are Y-shaped, and Y to T-shaped in hepatomegalia. The observations suggest that in hepatomegaly, the parenchyma pushes forward over the vessels and efferent bile ducts. The points of union or division penetrate deeper into the liver parenchyma, and their angles become more obtuse, approaching 180 degrees.

0696 COMPARISON OF ENDOSCOPIC RETROGRADE CHOLANGIOPANCREATOGRAPHY (ERCP) AND TRANSHEPATIC ANTEGRADE CHOLANGIOGRAPHY (THC) IN EVALUATING BILIARY STRICTURE. *(E.)* Wilkins, R.; Hatfield, A. (Northwick Park Hosp., Middlesex, England). *Digestion* 10(4/5):386-387, 1974.

0697 THE OPERATIVE CHOLANGIOGRAM: ITS INTERPRETATION, ACCURACY AND VALUE IN ASSOCIATION WITH CHOLECYSTECTOMY. *(E.)* McCormick, J. St. C.; Bremner, D. N.; Thomson, J. W. W.; McNair, T. J.; Philp, T. (R. Infirm., Edinburgh, Scotland). *Ann. Surg.* 180(6):902-906, 1974.

0698 ADENOCARCINOMA OF THE BILE DUCTS: RELATIONSHIP OF ANATOMICAL LOCATION TO CLINICAL FEATURES. *(E.)* Ingis, D. A.; Farmer, R. G. (Cleveland Clin. Found., Ohio). *Gut* 15(10):829-830, 1974.

0699 PAPILLOMA OF THE GALLBLADDER. REPORT OF A CASE. *(E.)* Kusakcioglu, O.; Aykam, T. B.; Kusakcioglu, A. (Beyoglu Insur. Hosp., Istanbul, Turkey). *Am. J. Gastroenterol.* 62(5):440-441, 1974.

0700 CLINICAL AND PATHOLOGICAL STUDY ON CHOLEDOCHOCYST. *(E.)* Suruga, K. (Juntendo Univ. Sch. Med., Tokyo, Japan). *Jpn. J. Surg.* 3(4):119-203, 1973.

0701 INTERMITTENT JAUNDICE DUE TO A CARCINOMA OF THE AMPULLA OF VATER. *(E.)* Phatak, P. S. (Whittington Hosp., London, England). *Proc. R. Soc. Med.* 67(10):1025-1026, 1974.

0702 CARBOHYDRATE TOLERENCE AND INSULIN RESPONSES IN OBSTRUCTIVE JAUNDICE. *(E.)* Soler, N. G.; Exon, P. D.; Paton, A. (Dudley Road Hosp., Birmingham, England). *Br. Med. J.* 4(5942):447-449, 1974.

0703 IMMUNOGLOBULINS IN OBSTRUCTIVE JAUNDICE. *(Cz.)* Fucikova, T.; Mikulenkova, M. (1st Clin. Intern. Med., Charles Univ., Prague, Czechoslovakia). *Sb. Lek.* 75(9):268-271, 1973.

0704 BIOCHEMICAL COMPOSITION OF THE BILE IN PATIENTS WITH CALCULOUS CHOLECYSTITIS. *(Rus.)* Galkin, V. A.; Borisov, V. G.; Maksimov, V. A.; Maslova, T. N.; Kasilova, R. Z.; Levchuk, V. F. (I.M. Sechenov 1st Moscow Med. Inst., USSR). *Ter. Arkh.* 46(4):45-48, 1974.

0705 NORMAL ANTIPYRINE METABOLISM IN PATIENTS WITH CHOLESTEROL CHOLELITHIASIS. EVIDENCE THAT THE DISEASE IS NOT DUE TO GENERALIZED HEPATIC MICROSOMAL DYSFUNCTION. *(E.)* Hepner, G. W.; Vesell, E. S. (Pennsylvania State Univ. Coll Med., Hershey). *Am. J. Dig. Dis.* 20(1):9-12, 1975.

0706 THE NON-OPERATIVE REMOVAL OF RETAINED COMMON DUCT STONES AFTER BILIARY SURGERY. *(E.)* Semb, B. K. H.; Norderval, Y.; Halvorsen, J. F. (Haukeland Hosp., Univ. Bergen, Norway). *Acta Chir. Scand.* 140(6):469-474, 1974.

0707 PREVALENCE OF GALLSTONES IN RELATION TO DIFFERING TREATMENT RATES FOR BILIARY DISEASE. *(E.)* Opit, L. J.; Greenhill, S. (Med. Sch., Univ. Birmingham, England). *Br. J. Prev. Soc. Med.* 28(4):268-272, 1974.

0708 THE PRESENT POSITION CONCERNING GALLSTONE DISSOLUTION. *(E.)* Bell, G. D. (St. James' Hosp. London, England). *Gut* 15(11):913-929, 1974.

0709 RATE OF IN VITRO INCORPORATION OF PRECURSORS INTO HEPATIC LIPIDS AND PROTEINS IN PATIENTS WITH EXTRAHEPATIC CHOLESTASIS. *(E.)* Stakeberg, H.; Lundborg, H.; Schersten, T. (Sahlgrenska Hosp., Univ. Goteborg, Sweden). *Eur. J. Clin. Invest.* 4(6):399-403, 1974.

0710 PRIMARY SCLEROSING CHOLANGITIS. *(E.)* Oviedo, M. A.; Volkmer, D.; Scanlon, E. F. (Northwest. Univ. Med. Sch., Evanston, Ill.). *Arch. Surg.* 109(6):747-749, 1974.

0711 DETERRANTS TO SUCCESS OF HEPATIC PORTICOENTEROSTOMY FOR BILIARY ATRESIA. *(E.)* Lilly, J. R.; Altman, R. P.; Randolph, J. G. (Univ. Colorado Med. Cent., Denver). *Gastroenterology* 67(4):810, 1974.

0712 A CASE OF TRUE DIVERTICULUM OF THE GALLBLADDER IN A DIABETIC WOMAN: ETIOLOGY AND DIAGNOSIS. *(Fr.)* Rached-Mohassel, M. A.; Handjani, A. A.; Nik-Akhtar, B.; Bagheri, F.; Teymourian, G. A. (Pahlavi Univ. Hosp. Cent., Teheran, Iran). *Bord. Med.* 6(13):1975-1980, 1973.

0713 THE EFFECT OF DECREASED GALLBLADDER STIMULATION ON ENTEROHEPATIC CYCLING AND KINETICS OF BILE ACIDS. *(E.)* Hepner, G. W. (Milton S. Hershey Med. Cent., Hershey, Pa.). *Gastroenterology* 67(4):797, 1974.

0714 VASCULAR INFLUENCES ON THE DYNAMIC STABILITY OF THE CHOLEDOCHODUODENAL JUNCTION. *(E.)* Tansy, M. F.; Innes, D. L.; Martin, J. S.; Kendall, F. M. (Temple Univ. Health Sci. Cent., Philadelphia, Pa.). *Am. J. Dig. Dis.* 19(12):1124-1137, 1974.

0715 PHARMACOLOGY OF CEPHALOTHIN IN THE BILIARY TRACT OF HUMANS. *(E.)* Mendelson, J.; Portnoy, J.; Sigman, H.; Dick, V. (Jewish Gen. Hosp., Montreal, Canada). *Antimicrob. Agents Chemother.* 6(6):659-665, 1974.

See also, 0034, 0118, 0335, 0366, 0384, 0385, 0440, 0460, 0524, 0545, 0572, 0573, 0581, 0589, 0591, 0592, 0594, 0651, 0671.

GENERAL

0716 THE LIVER IN KWASHIORKOR. *(E.)* Webber, B. L.; Freiman, I. (Dep. Pathol., Univ. Cape Town, South Africa). *Arch. Pathol.* 98(6):400-408, 1974.

A study to determine the correlation between the hepatomegaly of kwashiorkor and the pathologic changes at the cellular level was undertaken in 22 children (13 boys and 9 girls, 8 months-6 yr). All cases had the typical features of the disease; 14 children had abnormal irritability, lethargy, stupor or coma, but the other 8 were fully conscious. Clinical or biochemical jaundice appeared associated with more severe encephalopathy. Severe hypoglycemia was present in 3 children and 2 of these had seizures, which were considered a rather unusual feature. In the 6 cases with anemia, 2 had hemoglobin levels of 2.7 and 4.6 g/100 ml and exhibited purpura and subcutaneous hemorrhages. Serum protein levels were low in all cases (mean total protein 4.4 g/100 ml standard deviation 0.6 g/100 ml). Liver specimens from needle biopsy were fixed, and sections were cut to include parenchymal cells near portal tracts as well as near terminal hepatic veins. With light microscopy, all sections showed severe panlobular fatty metamorphosis, the cells being greatly distended by large lipid vacuoles which pushed the nuclei to a side; membranes between vacuoles and cell membranes appeared to be ruptured forming large cysts. Electron microscopy showed that hepatocytes in centrilobular and periportal areas exhibited similar large vacuoles in all cells with minimal electron density and without visible membranes, corresponding to the lipid vacuoles described above. Increase in size and number of lysosomes (thought to represent phospholipid) was noted in the hepatocytes, their highest concentration being around the bile canaliculi where they appeared to form an integral part of a group of organelles including many mitochondria, these last showing little abnormality. The nuclei exibited few abnormalities except being molded by the adjacent vacuoles and the nucleoli were larger then normal. Cell membranes appeared unremarkable. Three cases showed "alcoholic hyalin" (a finding which had been described before), and a mild increase of collagen adjacent to hepatocytes was noted, but did not appear to be of practical consequence. The changes in the liver parenchymal cells observed by electron microscopy were surprisingly mild and no relation with the degree of encephalopathy could be established.

0717 AN EXPERIMENTAL STUDY OF ACUTE NEONATAL ENTEROCOLITIS - THE IMPORTANCE OF BREAST MILK. *(E.)* Barlow, B.; Santulli, T. V.; Heird, W. C.; Pitt, J.; Blanc, W. A.; Schullinger, J. N. (Coll. Physicians Surg., Columbia Univ., New York, N.Y.). *J. Pediatr. Surg.* 9(5):587-595, 1974.

A model of necrotizing enterocolitis was designed using Sprague-Dawley rats. Rats were either fed with rat breast milk or with a formula which simulated rat milk and was composed of 6.0 g/100 ml protein, 10.0 g/100 ml fat, 9.6 g/100 ml carbohydrate, 1.2 g/100 ml ash and 152 calories/100 ml. Without hypoxia, all rats that were breast or formula fed, with or without *Klebsiella* contamination, remained healthy. When hypoxia was introduced, with or without contamination, all breast-fed animals remained healthy, while all formula-fed rats died at 2-5 days of age with preceding delayed gastric emptying, gaseous abdominal distention, diarrhea usually with occult or gross blood, lethargy, poor feeding, and cyanosis. The bowel was grossly necrotic and hemorrhagic at postmortem, and distended with gas with or without perforation and pneumoperitoneum. The intestines appeared normal at 12 and 24 hr with gaseous distention occurring at 36 hr, initially limited to the ileum and later involving the entire intestine. Histologically, the intestines of breast-fed animals were normal. Intestines of formula-fed rats showed dilatation with flattening of mucosal villi, then marked atrophy of villi with depletion of mucus in villous epithelium, and finally small ulcerations and bacterial invasion of the wall progressing in some to total necrosis. Stool cultures of breast-fed animals showed light or no growth of gram-negative organisms. Peritoneal and stool cultures of contaminated formula-fed rats had heavy *Klebsiella* growth, whereas those of the uncontaminated group showed heavy *Escherichia coli* growth. In rats subjected to daily hypoxia and *Klebsiella* contamination and fed either breast milk or formula for 24, 48, or 72 hr and then switched to the alternative feeding, all rats initially breast fed remained healthy. However, all rats fed formula for either 48 or 72 hr and then switched to breast milk died. In animals pretreated with immune globulin, all breast fed animals remained healthy and only 40% of the formula-fed rats developed the disease. Survivors had normal intestines at sacrifice. Stool samples had moderate growth of *E. coli*. In animals pretreated with Lactinex, disease incidence was also 40%; survivors had gaseous bowel distention and heavy fecal growth of *Klebsiella*. All rats pretreated with neomycin died. It was concluded that, through induction of passive enteric immunity and control of intestinal flora, breast milk may protect at-risk premature infants from acute enterocolitis.

0718 STUDY OF DYSPEPSIA IN A GENERAL PRACTICE AS ASSESSED BY ENDOSCOPY AND RADIOLOGY. *(E.)* Barnes, R. J.; Gear, M. W. L.; Nicol, A.; Dew, A. B. (Gloucestershire R. Hosp., Gloucester, Scotland). *Br. Med. J.* 4(5938):214-216, 1974.

A total of 50 patients (33 men and 17 women) presented with dyspepsia during a 6-month period and were studied by a combination of barium-meal examination and endoscopy. Each patient had suffered at least 1 of the following symptoms for more than 2 weeks: upper abdominal pain or discomfort related to food; retrosternal or upper abdominal pain related to posture; pain relieved by alkalis or vomiting; or pain experienced at night. On barium-meal examination 3 patients had gastric ulcers, 18 had pyloroduodenal disease, and 8 had hiatus hernia. The remaining 21 were normal. Endoscopic examination showed 30 patients with specific lesions of the esophagus, stomach, or duodenum. Three had gastric ulcers, none of which had been reported on barium-meal examination. One patient had a carcinomatous ulcer which had been reported on barium-meal exam as benign. Duodenal ulceration was noted in 11 patients and pyloroduodenal scarring without active ulceration in a further 12. In 4 patients in whom endoscopy showed pyloroduodenal deformity or ulceration, barium-meal examination had shown a hiatus hernia without any other lesion. There were also 2 patients with hiatus hernia and 1 with benign stricture of the esophagus with ulceration. Of the remaining 20 patients, 9 had inflammatory mucosal changes in the absence of specific lesions, and in 3 of these atrophic gastritis was found on histological examination. Endoscopic findings were normal in only 11. On cholecystography, 3 patients had gallstones and 3 a non-functioning gallbladder. Cholecystography failed in 1 patient, 1 had had a previous cholecystectomy, and function was normal in the remaining 42. Therefore, it is concluded that many patients with dyspepsia are receiving inadequate therapy, for underlying lesions have remained undiagnosed. It is suggested that endoscopy subsequent to the initial screening by a combined barium-meal examination with cholecystogram in selected patients will provide the most accurate diagnostic approach to the problem of dyspepsia.

0719 GASTROINTESTINAL DISORDERS. *(E.)* Paton, A. (Dudley Road Hosp., Birmingham, England). *Br. Med. J.* 4(5943):522-524, 1974.

0720 MEMORIAL LECTURE: THE PSYCHOSOMATIC APPROACH TO INDIVIDUAL SUSCEPTIBILITY TO DISEASE. *(E.)* Engel, G. L. (Univ. Rochester, Sch. Med. Dent., N.Y.). *Gastroenterology* 67(6):1085-1093, 1974.

0721 WATER, ELECTROLYTE AND ACID-BASE DISORDERS IN GASTROENTEROLOGY: THERAPEUTIC CONSIDERATIONS. *(E.)* Caprilli, R.; Carratu, R. (2nd Med. Clin., Univ. Rome, Italy). *Rend. Gastroenterol.* 6(2):121-131, 1974.

0722 CELLULAR IMMUNITY IN ENTERIC DISEASE. *(E.)* Collins, F. M.; Carter, P. B. (Trudeau Inst., Inc., Saranac Lake, N.Y.). *Am. J. Clin. Nutr.* 27(12):1424-1433, 1974.

0723 GASTROGENIC AND PANCREATOGENIC GASTROINTESTINAL INSUFFICIENCY. *(Ger.)* Zastrow, R. (Clin. Intern. Med., Univ. Rostock, Germany). *Z. Gesamte Inn. Med.* 29(22):936-940, 1974.

0724 EVOLUTION OF AND PROSPECTS FOR HEPATOLOGY AND GASTROENTEROLOGY. *(Fr.)* Bernier, J. J. (Lariboisiere Hosp., Paris, France). *Prog. Med.* 101(8):331-335, 1973.

0725 TREATMENT OF GASTROINTESTINAL INSUFFICIENCY WITH A COMBINATION OF A CHOLERETIC AND DIGESTIVE ENZYMES. *(It.)* Pietranera, P.; Rucci, R. (Dep. Med. Semeiotics, Univ. Parma, Italy). *Gaz. Med. Ital.* 132(10):517-520, 1973.

0726 CONGENITAL POSITIONAL ANOMALIES OF THE GASTROINTESTINAL TRACT AND THE EMBRYOLOGICAL MECHANISM INVOLVED: THREE CASES. *(Sp.)* Duaso Padin, R. (Concepcion Arenal Diagn. Treat. Cent., Santiago de Compostela, Spain). *Rev. Esp. Enferm. Apar. Dig.* 44(3):267-274, 1974.

0727 SUCROSE INTOLERANCE. *(Ger.)* Madzarovova-Nohejlova, J. (1st Med. Clin., Charles Univ., Plzen, Czechoslovakia). *Z. Gesamte Inn. Med.* 29(23):985-987, 1974.

0728 INTESTINAL, SALIVARY, AND SERUM IMMUNOGLOBULINS IN A GROUP OF CHILDREN WITH RECURRENT INTESTINAL AND RESPIRATORY INFECTIONS. *(Fr.)* Burnier, M. J.; Nussle, D.; Girard, J. P; Megevand, A. (no affil.). *Helv. Paediatr. Acta* 33(suppl.):25, 1974.

0729 COMPARISON OF BIOREACTIVE AND IMMUNOREACTIVE GASTRIN. *(E.)* Hansky, J; Royle, J. P.; Korman, M. G. (Dep. Med., Monash Univ., Melbourne, Australia). *Aust. J. Exp. Biol. Med. Sci.* 52(5): 841-846, 1974.

0730 CORRELATION BETWEEN ACID AND PEPSIN SECRETION OF THE STOMACH UNDER PHYSIOLOGICAL AND PATHOLOGICAL CONDITIONS. *(E.)* Bonfils, S. (Bichat Hosp., Paris, France). *Rend. Gastroenterol.* 6(1): 65-73, 1974.

0731 TREATMENT OF DYSPEPTIC SYNDROMES WITH DISKILENE. *(Sp.)* Garcia Perez, M. (Hermanos Aznar Outpatient Clin., Madrid, Spain). *Rev. Esp. Enferm. Apar. Dig.* 44(2):187-192, 1974.

0732 PARENTERAL HYPERALIMENTATION - FIVE YEARS' CLINICAL EXPERIENCE. *(E.)* Myers, R. N.; Smink, Jr., R. D.; Goldstein, F. (Lankenau Hosp., Philadelphia, Pa.). *Am. J. Gastroenterol.* 62(4): 313-324, 1974.

0733 THE RADIOLOGIC MANIFESTATIONS OF HENOCH-SCHOENLEIN PURPURA. *(E.)* Macpherson, R. I. (Child. Hosp., Winnipeg, Canada). *J. Can. Assoc. Radiol.* 25(4):275-278, 1974.

0734 EXPLORATORY CELIOTOMY FOR ACUTE ABDOMINAL PAIN. *(E.)* Yajko, R. D.; Steele, Jr., G. (Univ. Colorado Med. Cent., Denver). *Am. J. Surg.* 128(6):773-776, 1974.

0735 HERIDITARY HEMORRHAGIC TELEANGIECTASIS (RENDU-WEBER-OSLER'S DISEASE) WITH INTESTINAL INVOLVEMENT SUCCESSFULLY TREATED BY SURGERY. *(E.)* Bruusgaard, A.; Juhl, E. (Frederiksberg Hosp., Copenhagen, Denmark). *Gastroenterology* 67(5):1001-1005, 1974.

0736 FAMILIAL POLYPOSIS OF THE GASTROINTESTINAL TRACT. *(E.)* Utsunomiya, J. (Tokyo Med. Dent. Univ., Japan). *Asian Med. J.* 17(2):61-64, 1974.

0737 EPIDEMIOLOGY OF GASTROINTESTINAL CANCER. *(Fr.)* Audigier, J. C.; Lambert, R. (Edouard Herriot Hosp., Lyon, France). *Arch. Fr. Mal. App. Dig.* 63:413-432, 1974.

0738 TUMORS OF THE DIGESTIVE TRACT AND LIVER. *(Fr.)* Schlumberger, J. P.; Cathala, G. (no affil.). *Gaz. Hop.* 145(13):598-599, 1973.

0739 ISCHEMIC GASTROINTESTINAL LESIONS IN THE NEONATE. *(E.)* Touloukian, R. J. (Yale Univ. Sch. Med., New Haven, Conn.). *Paediatrician* 3(3-5):230-236, 1974.

0740 KAPOSI'S SARCOMA PRESENTING WITH DIARRHEA AND PROTEIN-LOSING ENTEROPATHY. *(E.)* Novis, B. H.; King, H.; Bank, S. (Groote Schuur Hosp., Cape Town, South Africa). *Gastroenterology* 67(5):996-1000, 1974.

0741 PATHOMORPHOLOGIC, BIOCHEMICAL, AND DIAGNOSTIC ASPECTS OF GASTRINOMAS (ZOLLINGER-ELLISON SYNDROME). *(E.)* Creutzfeldt, W.; Arnold, R.; Creutzfeldt, C.; Track, N. S. (Dep. Med., Univ. Gottingen, Germany). *Hum. Pathol.* 6(1):47-76, 1975.

0742 METIAMIDE IN ZOLLINGER-ELLISON SYNDROME. *(E.)* Thompson, M. H.; Venables, C. W.; Miller, I. T.; Reed, J. D.; Sanders, D. J.; Grund, E. R.; Blair, E. L. (Univ. Newcastle upon Tyne, England). *Lancet* 1(7897):35-36, 1975.

0743 VALUE AND LIMITATIONS OF DETERMINING BLOOD GASTRIN IN THE DIAGNOSIS OF ZOLLINGER-ELLISON'S SYNDROME. *(Fr.)* Bernades, P.; Bonfils, S. (Bichat Hosp., Paris, France). *Arch. Fr. Mal. App. Dig.* 63(4):265-268, 1974.

0744 GASTROINTESTINAL BLEEDING: MEDICAL ASPECTS. *(Ger.)* Schuster, H. P. (2nd Med. Clin., Univ. Mainz, Germany). *Langenbecks Arch. Chir.* 337: 511-518, 1974.

0745 LOCAL HEMOSTASIS IN CONSERVATIVE TREATMENT OF GASTROINTESTINAL BLEEDING. *(Ser.)* Prcic, M. (Surg. Clin., Sarajevo, Yugoslavia). *Med. Arh.* 27(4):309-311, 1973.

0746 UPPER GASTROINTESTINAL BLEEDING. *(E.)* Patterson, M. (Univ. Texas Med. Branch, Galveston). *J. Miss. State Med. Assoc.* 15(11): 469-475, 1974.

0747 HEMORRHAGE OF THE UPPER GASTROINTESTINAL TRACT IN HEMATOLOGICAL DISEASES. *(Sp.)* Perez Carnero, A.; Montero Garcia, J. M.; Muro, J.; Hita Perez, J.; Cano Lopez, J. M. (La Paz Soc. Welfare Polyclin., Free Univ., Madrid, Spain). *Rev. Esp. Enferm. Apar. Dig.* 44(2):141-156, 1974.

0748 RADIOLOGICAL DIAGNOSIS OF BLEEDING FROM THE ESOPHAGUS, STOMACH, OR DUODENUM. *(Ger.)* Bucheler, E. (Radiol. Clin., Univ. Bonn, Germany). *Langenbecks Arch. Chir.* 337:497-503, 1974.

0749 GASTROINTESTINAL BLEEDING (ESOPHAGUS, STOMACH, DUODENUM) IN INFANTS AND CHILDREN. *(Ger.)* Joppich, I. (Mannheim Clin., Univ. Heidelberg, Germany). *Langenbecks Arch. Chir.* 337:547-550, 1974.

0750 EOSINOPHILIC GASTROENTERITIS, GLUTEN ENTEROPATHY, AND DERMATITIS HERPETIFORMIS. *(E.)* Bennett, R. A.; Whitelock, III, T.; Kelley, Jr., J. L. (Donald N. Sharp Mem. Community Hosp., San Diego, Calif.). *Am. J. Dig. Dis.* 19(2):1154-1161, 1974.

0751 ACUTE VIRAL GASTROENTERITIS. *(E.)* Kapikian, A. Z. (Natl. Inst. Allergy Infect. Dis., Bethesda, Md.). *Prev. Med.* 3(4):535-542, 1974.

0752 THE MANAGEMENT OF ACUTE DIARRHEA IN INFANTS. *(Fr.)* Moreno, J. L. (no affil.). *Gaz. Med. France* 80(37):6147-6156, 1973.

0753 DEFINITION AND DISTINGUISHING CRITERIA OF DIARRHEA. *(Ger.)* Koelsch, K. A. (Magdeburg-Altstadt Reg. Hosp., Germany). *Z. Gesamte Inn. Med.* 29(14):591-594, 1974.

0754 ACUTE DIARRHEA IN A RESIDENTIAL INSTITUTION FOR THE RETARDED: USEFULNESS OF FECAL LEUKOCYTE EXAMINATION. *(E.)* Pierce, J. E.; DuPont, H. L.; Lewis, K. R. (Univ. Texas Health Sci. Cent., Houston). *Am. J. Dis. Child.* 128(6):772-775, 1974.

0755 DIARRHEA ASSOCIATED WITH RIGHT HEMICOLECTOMY: PHYSIOPATHOLOGICAL CONCEPTS AND THERAPEUTIC DEDUCTIONS BASED ON 6 CASES. *(Fr.)* Gendre, J. P.; Le Quintrec, Y. (Albert Chenevier Hosp., Creteil, France). *Nouv. Presse Med.* 3(34):2149-2152, 1974.

0756 PATHOPHYSIOLOGY OF THE DIARRHEA ASSOCIATED WITH CHOLERA. *(Fr.)* Thomas, J.; Josserand, C.; Andre, L. J. (A. N. P. Cent. Hosp., Algiers, Algeria). *Arch. Fr. Mal. App. Dig.* 63(4):327-341, 1974.

0757 CHOLERA. *(E.)* Carpenter, C. C. J. (Univ. Hosp., Cleveland, Ohio). *Prev. Med.* 3(4): 456-460, 1974.

0758 COMPARISON OF THE EFFECTS OF ENTEROTOXINS OF Shigella dysenteriae AND Vibrio cholerae ON THE ADENYLATE CYCLASE SYSTEM OF THE RABBIT INTESTINE. *(E.)* Flores, J.; Grady, G. F.; McIver, J.; Witkum, P.; Beckman, B.; Sharp, G. W. G. (Massachusetts Gen. Hosp., Boston). *J. Infect. Dis.* 130(4): 374-379, 1974.

0759 RESPONSE OF MAN TO INFECTION WITH Vibrio cholerae. II. PROTECTION FROM ILLNESS AFFORDED BY PREVIOUS DISEASE AND VACCINE. *(E.)* Cash, R. A.; Music, S. I.; Libonati, J. P.; Craig, J. P.; Pierce, N. F.; Hornick, R. B. (Univ. Maryland Sch. Med., Baltimore). *J. Infect. Dis.* 130(4):325-333, 1974.

0760 ROLE OF HOUSEHOLD ANIMALS IN MAINTENANCE OF CHOLERA INFECTION IN A COMMUNITY. *(E.)* Sanyal, S. C.; Singh, S. J.; Tiwari, I. C.; Sen, P. C.; Marwah, S. M.; Hazarika, U. R.; Singh, H.; Shimada, T.; Sakazaki, R. (Inst. Med. Sci., Banaras Hindu Univ., Varanasi, India). *J. Infect. Dis.* 130(6):575-579, 1974.

0761 RESISTENCE OF Vibrio parahaemolyticus AND Vibrio cholera TO HOSTILE ENVIRONMENTAL CONDITIONS. *(It.)* Campanini, M.; Zupan, J.; Paini, L.; Vicini, E. (Exp. Stn. Food Preserv. Ind., Parma, Italy). *Ind. Conserve* 49(3):170-172, 1974.

0762 CHOLERA-LIKE ENTERITIS ASSOCIATED WITH ENTEROTOXIGENIC BACTERIA. *(Gr.)* Arseni, A. (no affil.). *Iatriki* 24(2):89-96, 1973.

0763 GASTROENTERITIS DUE TO NON-AGGLUTINABLE (NON-CHOLERA) VIBRIOS. *(E.)* Dakin, W. P. H.; Howell, D. J.; Sutton, R. G. A.; O'Keefe, M. F.; Thomas, P. (Med. Dep., Qantas Airways Ltd., Sydney, Australia). *Med. J. Aust.* 2(61):487-490, 1974.

0764 CHOLERA-LIKE TOXIC EFFECT OF CULTURE FILTRATES OF Escherichia coli. *(E.)* Nalin, D. R.; Bhattacharjee, A. K.; Richardson, S. H. (Bowman Gray Sch. Med., Wake Forest Univ., Winston-Salem, N.C.). *J. Infect. Dis.* 130(6):595-601, 1974.

0765 ENTEROPATHOGENIC Escherichia coli INFECTION IN A NEWBORN NURSERY. *(E.)* Kaslow, R. A.; Taylor, Jr., A.; Dweck, H. S.; Bobo, R. A.; Steele, C. D.; Cassady, Jr., G. (Cent. Dis. Control, Atlanta, Ga.). *Am. J. Dis. Child.* 128(6):797-801, 1974.

0766 IN VIVO PRODUCTION AND INACTIVATION OF Escherichia coli ENTEROTOXIN. *(E.)* Donta, S. T.; Moon, H. W.; Whipp, S. C.; Skartvedt, S. M. (Dep. Med., Univ. Iowa, Iowa City). *Gastroenterology* 67(5):983-990, 1974.

0767 THE EFFECT OF IMMUNIZATION WITH A ROUGH STRAIN OF Salmonella enteritidis ON THE FATE OF Salmonella typhimunium IN MICE. *(E.)* Davies, R.; Kotlarski, I. (Dep. Microbiol., Univ. Adelaide, Australia). *Aust. J. Exp. Biol. Med. Sci.* 52(5):779-789, 1974.

0768 THE VALUE OF TRIMETHOPRIM-SULFAMETHOXAZOLE IN THE TREATMENT OF SALMONELLA INFECTIONS. *(E.)* Lurie, I.; Felner, S.; Burke, M. (Ichilov Munic. Gov. Hosp., Tel Aviv, Israel). *Isr. J. Med. Sci.* 10(11):1420-1424, 1974.

0769 CURRENT INCIDENCE OF INTESTINAL SALMONELLOSIS IN A CHILDREN'S HOSPITAL IN PARIS. *(Fr.)* Laplane, R.; Begue, P.; Omanga, U.; Rizo, O. (no affil.). *Bull. Acad. Natl. Med. (Paris)* 158(3):238-241, 1974.

0770 EPIDEMIOLOGY OF Salmonella Typhi INFECTION IN A MIGRANT LABOR CAMP IN DADE COUNTY, FLORIDA. *(E.)* Feldman, R. E.; Baine, W. B.; Nitzkin, J. L.; Saslaw, M. S.; Pollard, Jr., R. A. (Dade Cty. Dep. Public Health, Miami, Fla.). *J. Infect. Dis.* 130(4):334-342, 1974.

0771 IMMUNOLOGICAL STUDY OF TYPHOID: IMMUNOGLOBINS, C_3, ANTIBODIES, AND LEUKOCYTE MIGRATION INHIBITION IN PATIENTS WITH TYPHOID FEVER AND TAB-VACCINATED INDIVIDUALS. *(E.)* Kumar, R.; Malaviya, A. N.; Murthy, R. G. S.; Venkataraman, M.; Mohapatra, L. N. (All-India Inst. Med. Sci., New Delhi). *Infect. Immun.* 10(6):1219-1225, 1974.

0772 THE RELATION BETWEEN INSULIN SECRETION GLUCOSE TOLERANCE, GROWTH HORMONE, AND SERUM PROTEINS IN PROTEIN-CALORIE MALNUTRITION. *(E.)* Becker, D. J.; Pimstone, B. L.; Hansen, J. D. L. (Dep. Pediatr. Child Health, Univ. Cape Town, South Africa). *Pediatr. Res.* 9(1):35-39, 1974.

0773 PATTERNS OF PROTEIN-ENERGY MALNUTRITION IN EARLY CHILDHOOD IN JORDAN. *(E.)* Hijazi, S. S. (Ashragieh Hosp., Amman, Jordan). *Am. J. Clin. Nutr.* 27(11):1254-1258, 1974.

0774 EFFECT OF MALNUTRITION ON COLONIC HEALING. *(E.)* Irvin, T. T.; Hunt, T. K. (Dep. Surg., Univ. Leeds, England). *Ann. Surg.* 180(5):765-772, 1974.

0775 BASAL CELL ADENOMA OF THE SALIVARY GLANDS. *(E.)* Thawley, S. E.; Ward, S. P.; Ogura, J. H. (Washington Univ. Sch. Med., St. Louis, Mo.). *Laryngoscope* 84(10):1756-1766, 1974.

0776 AN ULTRASTRUCTURAL STUDY OF ADENOID CYSTIC CARCINOMA OF THE HUMAN SALIVARY GLAND. *(E.)* Panicker, K. N. S.; Potdar, G. G.; Sirsat, S. M. (Cancer Res. Inst., Bombay, India). *Indian J. Cancer* 11(2):168-176, 1974.

0777 BENIGN LYMPHOEPITHELIAL LESIONS OF THE SALIVARY GLANDS. *(E.)* Kelly, D. R.; Spiegel, J. C.; Maves, M. (Ohio State Univ. Coll. Med., Columbus). *Arch. Otolaryngol.* 101(1):71-75, 1975.

0778 HEPATIC INVOLVEMENT IN SJOGREN'S SYNDROME. *(E.)* Lopes, J. D.; Acras, S.; Borini, P.; De Moraes-Filho, J. P. P.; Bettarello, A. (Sao Paulo Med. Sch., Univ. Sao Paulo, Brazil). *Acta Gastroenterol. Latinoam.* 6(1):1-5, 1974.

0779 PAROTIDITIS DURING CAT SCRATCH DISEASE. *(Fr.)* Laudenbach, P.; Harar, E.; Hallard, M.; Gros, F. (Corentin-Celton Hosp., Issy-les-Moulineaux, France). *Nouv. Presse Med.* 3(28):1753-1754, 1974.

See also, 0341, 0342, 0344, 0345, 0346, 0348, 0370, 0374, 0438.

GENERAL

Parasitology

0780 GASTROINTESTINAL PROTEIN LOSS IN HOOKWORM AND ROUNDWORM INFECTIONS. *(E.)* Gupta, M. C.; Basu, A. K.; Tandon, B. N. (All India Inst. Med. Sci., New Delhi). *Am. J. Clin. Nutr.* 27:1386-1389, 1974.

Gastrointestinal protein loss was studied using ^{51}Cr-albumin in 11 patients (10 males and 1 female, aged 15-16 yr) having mild hookworm and/or roundworm infection. Of these, 7 had pure hookworm infection and 4 had combined infection. The dietary history

was not investigated in detail; all consumed mainly a cereal-legume based diet with minimal quantities of milk and other animal protein. All patients were hospitalized for the study and given a diet of about 2,200 kcal, 50 g protein and 100 g fat/day with added vitamins. The main clinical presentation was asthenia, fatigue, vague abdominal pain, and bowel disturbance characterized by incomplete evacuation with or without passage of unformed stools. Only mild infection was noted with ova counts ranging from 33-1,340 eggs/g for hookworm and 29-983 eggs/g for roundworm. Gastrointestinal protein loss in terms of 96-hr fecal excretion of ^{51}Cr was less than 0.75% in 2 control subjects. All except 1 patient had gastrointestinal protein loss below the upper limit of normal. The 1 patient with increased gastrointestinal protein loss had pure hookworm infection; this patient also had the highest ova count in the series. No difference was noted regarding gastrointestinal protein loss between pure hookworm infection and mixed hookworm and roundworm infection. Derangement of absorption of D-xylose was present in 3 of the 7 patients with hookworm infection and in 2 of the 4 patients with mixed hookworm and roundworm infections. It is concluded that the malabsorption recorded in patients with hookworm disease is related to associated protein malnutrition rather than worm infection.

0781 Schistosomiasis mansoni IN YEMENI IN CALIFORNIA: DURATION OF INFECTION, PRESENCE OF DISEASE, THERAPEUTIC MANAGEMENT. *(E.)* Warren, K. S.; Mahmound, A. A. F.; Cummings, P. Murphy, D. J.; Houser, H. B. (Case Western Reserve Univ. Sch. Med., Cleveland, Ohio). *Am. J. Trop. Med. Hyg.* 23(5):902-909, 1974.

Of 218 Yemeni agricultural workers in the San Joaquin Valley of California, 122 (56%) had *Schistosoma mansoni* eggs in their stools. Quantitative egg counts with the Kato thick smear method showed that 57% of those infected had light infections (1-100 eggs/g), 27% moderate infections (101-400 eggs/g), and 16% heavy infections (>400 eggs/g; mean 918 eggs/g). The time away from Yemen varied from 2 months to 20 yr, a period in which the prevalence of schistosomiasis had remained constant in Yemen. In those away for less than 5 yr the prevalence of schistosomiasis was 59% with a mean egg output of 236 eggs/g; in those away for more than 5 yr the prevalence was 32% with a mean egg output of 65 egg/g, despite the fact that 75% of the latter had returned to Yemen for short visits. Results of Fisher's exact probability test showed that the lower egg output in those away from Yemen more than 5 yr was statistically significant. On the basis of these findings the mean life span of the Yemeni strain of *S. mansoni* in man was estimated to be 5-10 yr. There were no differences in symptoms, such as weakness, diarrhea, and abdominal pain, between the uninfected proportion of the population, total infected group, and the heavily infected subgroup. None of the patients had hepatomegaly and only 2 had splenomegaly (1 lightly and the other heavily infected). Because of the toxicity of antischistosomal drugs and the lack of treatment facilities, only the 22 most heavily infected patients were treated with niridazole (12.5 mg/kg twice/day for 7 days, p.o.). Side effects (e.g., headache, 64%; nausea and/or vomiting, 67%; dizziness, 41%) were common with niridazole but were not severe. Mean egg production in 9 workers followed for 3-7 months was reduced by 97%. The 2 patients with splenomegaly were given stibocaptate (200 mg/week for 5 weeks, i.m.).

0782 A CONTROLLED STUDY OF MORBIDITY OF Schistosomiasis mansoni IN ST. LUCIAN CHILDREN, BASED ON QUANTITATIVE EGG EXCRETION. *(E.)* Cook, J. A.; Baker, S. T.; Warren, K. S.; Jordan, P. (Res. Control Dep., Minist. Health, Castries, St. Lucia, West Indies). *Am. J. Trop. Med. Hyg.* 23(4):625-633, 1974.

Morbidity due to schistosomiasis was studied among 75 St. Lucian school children (aged 7-16 yr) who were heavily infected (400 or more eggs/ml feces), moderately infected (100-300 eggs/ml), or lightly infected (10-75 eggs/ml) with *Schistosomiasis mansoni*; 56 uninfected children served as controls. Among the uninfected group, 32 were found during the study to have become lightly infected. The groups were comparable with respect to sex, the heavily infected children being somewhat older than the uninfected children. Gastrointestinal symptoms were relatively frequent among all groups, the frequency being independent of infection with *S. mansoni*. The groups did not differ in terms of anthropometric measurements. Hepatomegaly and splenomegaly were significantly more frequent in the heavy-moderate infection groups, and extension of the liver below the costal margin increased with the intensity of infection. The serum globulin level and degree of skin-test reactivity were directly related to the intensity of infection, while the serum albumin level was inversely related. Infection with *S. mansoni* did not result in disease in most of the children investigated.

0783 PLEURAL EMPYEMA DUE TO RUPTURED AMOEBIC LIVER ABSCESS. *(E.)* Rasaretnam, R.; Paul, A. T. S.; Yoganathan, M. (Colombo Gen. Hosp., Ceylon). *Br. J. Surg.* 61(9):713-715, 1974.

The clinical features and treatment regimens of 31 patients with empyema secondary to ruptured amebic liver abscess are presented. The diagnosis was initially established by chest x-ray and was confirmed in each case by aspiration of typical "chocolate sauce" pus from the pleural cavity. The empyema fluid was positive for amebae in only 1 case. Treatment consisted of pleural drainage by intercostal catheter in 27 patients, 3 of whom died due to poor general condition. Fourteen subsequently underwent decortication; 1 death in this group was of a severely jaundiced patient. Another 3 underwent primary decortication while the final patient died suddenly during aspiration, possibly due to a reaction to the local anesthetic. All but 2 patients were male. Symptoms, which included chest pain, fever, cough, and abdominal pain, were less than 4 weeks' duration in 18 of the 31 patients. All had symptoms of pleural effusion and 15 had intercostal tenderness and 14 had hepatomegaly. Only 2 patients

were jaundiced; 15 had anemia (Hgb < 10 gm%). Mean white cell count was 15,333/ml and erythrocyte sedimentation rate was elevated in 24 of 25 patients tested. Secondary infection with pyogenic organisms occurred in one-third of the cases.

0784 PARASITOLOGIC INVESTIGATIONS IN GASTROENTEROLOGIC PRACTICE. *(E.)* Mantell, C. A.; Yoeli, M. (St. Vincent's Med. Cent., New York, N.Y.). *NY State J. Med.* 75(1):39-43, 1975.

0785 PROTOZOAL DISEASES ENDEMIC IN THE UNITED STATES. THE TEACHING OF PARASITOLOGY AS DETERMINED BY ENDEMIC PROTOZOAL DISEASES. *(E.)* Neva, F. A. (Natl. Inst. Allergy Infect Dis., Bethesda, Md.). *Am. J. Trop. Med. Hyg.* 23(4):731-735, 1974.

0786 GIARDIASIS: A COMMON CAUSE OF PROLONGED DIARRHOEA IN ADULTS. *(E.)* Heap, T. (North Shore Med. Cent., St Leonards, Australia). *Med. J. Aust.* 2(16):592-595, 1974.

0787 IMMUNODIAGNOSTIC TESTS FOR BILHARZIASIS. II. REACTIVITY OF VARIOUS SCHISTOSOME ANTIGENS IN DIFFERENT SEROLOGICAL TESTS. *(E.)* Umaly, R. C.; Oelerich, S., Haas, J. (Bernard-Nocht Inst. Ship, Trop. Dis., Hamburg, Germany). *Tropenmed. Parasitol.* 25(4):413-421, 1974.

0788 IMMUNODIAGNOSTIC TESTS FOR BILHARZIASIS. III. SERODIAGNOSIS OF Schistosoma haemotobium INFECTION AND INFLUENCE OF CHEMOTHERAPY ON IMMUNODIAGNOSTIC TESTS. *(E.)* Umaly, R. C.; Oelerich, S.; Haas, J. (Bernard-Nocht Inst. Ship, Trop. Dis., Hamburg, Germany). *Tropenmed. Parasitol.* 25(4):422-432, 1974.

0789 EXPULSION OF Nippostrongylus brasiliensis FROM THE INTESTINE OF RATS: ATTEMPTS TO INITIATE WORM EXPULSION BY CELL TRANSFER IN AN IMMUNOLOGICALLY INERT ALLOGENEIC ENVIRONMENT. *(E.)* Kelly, J. D.; Love, R. J.; Dineen, J. K. (Dept. Vet. Pathol., Univ. Sydney, Australia). *Int. Arch. Allergy Appl. Immunol.* 46(6):880-893, 1974.

0790 NECROTIC AMEBIC COLITIS. *(E.)* Cutler, D.; Avendano, E.; Maldonado, P.; Acosta, M. (Fac. Med., Natl. Auton. Univ., Mexico City, Mexico). *Am. J. Gastroenterol.* 62(4):345-349, 1974.

0791 INTESTINAL AMEBIASIS. RADIOLOGICAL AND SURGICAL ASPECTS. *(E.)* Balikian, J. P.; Nahra, K. S.; Uthman, S. M. (Dep. Radiol., Am. Univ. Beirut, Lebanon). *J. Med. Liban.* 27(4):413-423, 1974.

0792 TINIDAZOLE IN SYMPTOMATIC INTESTINAL AMOEBIASIS. *(E.)* Prakash, C.; Bansal, B. C.; Bansal, M. R. (Med. Coll., Rohtak, India). *J. Trop. Med. Hyg.* 77(7):165-167, 1974.

0793 COMPARATIVE TRIAL OF TINIDAZOLE AND METRONIDAZOLE IN INTESTINAL AMEBIASIS. *(E.)* Misra, N. P.; Laiq, S. M. (Gandhi Med. Coll., Bhopal, India). *Curr. Ther. Res.* 16(12):1255-1263, 1974.

0794 THE USE OF MEBENDAZOLE AS A BROAD-SPECTRUM ANTHELMINTIC IN RHODESIA. *(E.)* Goldsmid, J. M. (Dep. Med. Microbiol., Univ. Rhodesia, Salisbury). *S. Afr. Med. J.* 48(54):2265-2266, 1974.

0795 ANTHELMINTIC ACTIVITY OF MEBENDAZOLE AGAINST Cysticercus fasciolaris. *(E.)* Thienpont, D.; Vanparijs, O.; Hermans, L. (Dep. Chemother., Janssen Pharm., Beerse, Belgium). *J. Parisitol.* 60(6):1052-1053, 1974.

0796 CASE REPORT. AMOEBIC PERICARDITIS. *(E.)* Faerber, E. N.; Segal, F.; Sischy, N. L. (Baragwanath Hosp., Johannesburg, South Africa). *Br. J. Radiol.* 47(563):816-818, 1974.

0797 HEPATIC AMEBIASIS IN 1973. *(Fr.)* Cenac, A. (Paris Hosp., France). *Gaz. Med. France* 80(34):5561-5576, 1973.

0798 REPEATED ANTICHOLERA VACCINATIONS AND THEIR IMMUNOSUPPRESSIVE EFFECT LEADING TO THE DEVELOPMENT OF HEPATIC AMEBIASIS. *(Fr.)* Bastin, R.; Charmot, G.; Calamy, G.; Baudet, M. (Claude Bernard Hosp., Paris, France). *Ann. Med. Interne* 125(6/7): 513-518, 1974.

0799 SURGICAL TREATMENT OF PARASITIC DISEASES OF THE LIVER. *(Fr.)* Rohner, A. (Surg. Clin., Univ. Geneva, Switzerland). *Helv. Chir Acta* 41(5/6): 665-674, 1974.

See also, 0211, 0378, 0567, 0601, 0762.

0800 AN EVALUATION OF THE ROSETTE INHIBITION TEST IN CROHN'S DISEASE. *(E.)* Clarke, J. R.; Richens, E. R.; Williams, M. J.; Gough, K. R.; Ancill, R. J. (R. United Hosp. (North), Bath, England). *Gut* 15(9):725-727, 1974.

Serial rosette inhibition tests were performed on 9 patients (4 females and 5 males, aged 16-71 yr) with Crohn's disease lasting from 1-22 yr, who were undergoing immunosuppressive therapy. In 23 normal subjects tested, the mean minimum inhibitory concentration for the group was 24.3. Age slightly affected the endpoint; sex did not. When phosphate-buffered saline (pH 7.35) was used in the incubation medium (30 cases), the percentage of rosette-forming cells was 25.1. When Hank's solution was used (37 cases) this number fell to 11.2. However, the minimum inhibitory concentration was not affected by the percentage of rosette-forming cells. The minimum inhibitory concentration was 24.8 with phosphate-buffered saline and 24.1 with Hank's solution. When not on immunosuppression therapy, the patients in an active phase of the disease showed a significantly lower minimum inhibitory concentration than those patients with inactive disease who gave results close to the normal range. After a short period on immunosuppression, high minimum inhibitory concentration values were obtained in the patients tested, all of whom remained in an active phase at this time. This rise in minimum inhibitory concentration became more marked with increasing length of immunosuppression. After more than 4 days treatment, no significant difference in minimum inhibitory concentration was apparent between the active and inactive groups. Immunosuppression appears to be the critical factor affecting the minimum inhibitory concentration. However, despite the fact that the test clearly differentiates between groups of patients receiving immunosuppression and those not so treated, this differentiation in itself is not sufficient to be of practical value in controlling immunosuppressive dosage in Crohn's disease. There was no consistent correlation with the degree of disease activity.

0801 NO EVIDENCE FOR DECREASED LYMPHOCYTE REACTIVITY IN CROHN'S DISEASE. *(E.)* Bird, A. G.; Britton, S. (Div. Immunobiol., Karolinska Inst., Stockholm, Sweden). *Gastroenterology* 67(5): 926-932, 1974.

The immunological status was investigated in a selected series of 20 patients (14 women and 6 men, average age 32.2 yr) with active Crohn's disease, ranging in duration from 3 months to 8 yr. Patients were not on corticosteroids for at least 2 months prior to the study. Two patients were on adrenocorticotrophic hormone; the majority were taking salicylazosulfapyridine. *In vitro* lymphocyte proliferative response of Crohn's disease patients to phytohemagglutinin and to purified protein derivative tuberculin did not differ significantly from that of 15 enteritis patients or from 15 healthy controls. Thus there is no evidence for a generalized reduction in T lymphocyte responsiveness in Crohn's disease. In studying the lymphocytes in the lymph nodes draining diseased bowel, mesenteric lymph nodes were removed at bowel resection on 6 patients. Again no evidence of impaired T-cell responsiveness could be found in these nodal lymphocytes as compared with the circulating lymphocyte activity in the same patients or to that of the control groups. The proportion of lymphocytes forming rosettes with sheep erythrocytes was similar to controls in the peripheral blood specimens studied. The mesenteric lymph node cell preparations also contained a high percentage of T-cells not different from control nodes. Mantoux reactivity was tested in 12 of the patients. Of the 12, 7 were nonreactive as compared with 2 of 14 enteritis controls. However, there was no correlation between Mantoux negativity and the *in vitro* lymphocyte reactivity to the antigens. It is possible that the disease process was interfering with the efferent limb of the skin hypersensitivity response, perhaps with the inflammatory component. Only 1 patient showed a markedly reduced *in vitro* lymphocyte responsiveness to both mitogens and antigens. She was Mantoux-negative and showed signs of severe malnutrition, supporting the concept of a link between malnutrition and reduced cell-mediated immunity. It is suggested that the case of phytohemagglutinin lymphocyte hyporesponsiveness described in Crohn's disease may be accounted for by prolonged malabsorption and malnutrition in some patients rather than to a uniform depression of T lymphocyte immunity.

0802 CHANGING PATTERNS IN EPIDEMIOLOGY OF CROHN'S DISEASE. *(E.)* Miller, D. S.; Keighley, A. C.; Langman, M. J. S. (Med. Sch., Nottingham Univ., England). *Lancet* 2(7882):691-693, 1974.

0803 GASTRIC ANTRAL NARROWING IN CHRONIC GRANULOMATOUS DISEASE OF CHILDHOOD. *(E.)* Griscom, N. T.; Kirkpatrick, Jr., J. A.; Girdany, B. R.; Berdon, W. E.; Grand, R. I.; Machie, G. G. (Harvard Med. Sch., Boston, Mass.). *Pediatrics* 54(4):458-460, 1974.

0804 INFLAMMATORY BOWEL DISEASE. THE SURGICAL PATHOLOGY OF CROHN'S DISEASE AND ULCERATIVE COLITIS. *(E.)* Price, A. B.; Path, M. R. C.; Morson, B. C.; Path, F. C. (St. Mark's Hosp., London, England). *Hum. Pathol.* 6(1):7-29, 1975.

See also, 0451, 0492.

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PRE-CLINICAL SCIENCES

MORPHOLOGY, CELLULAR STRUCTURE AND HISTOCHEMISTRY

0805 QUANTITATIVE HISTOLOGY OF THE GASTRIC MUCOSA: MAN, DOG, CAT, GUINEA PIG, AND FROG. *(E.)* Hogben, C. A. M.; Kent, T. H.; Woodward, P. A.; Sill, A. J. (Dep. Physiol. Biophys., Univ. Iowa, Iowa City). *Gastroenterology* 67(6):1143-1154, 1974.

A simpler method than those previously reported was applied to the quantitative histology of the gastric mucosae from man, dog, cat, guinea pig, and frog. Standardized strips of stomach wall, which varied according to the anatomy of the species, were selected for histological processing. After routine paraffin processing, sections were cut at 5 μ and stained in succession with 0.5% aldehyde fuchsin in 70% alcohol, with the usual steps for the periodic acid-Schiff technique, with Harris hematoxylin and with aurantia. One color 35-mm photomicrograph was taken from various sites in the corpus mucosa and magnified from 1000 to 1500. The various cell types were counted and from these values the percentage of each component calculated. Fourteen sites in man, 11 sites in dogs and cats, 8 sites in guinea pigs, and 3 sites in frogs were considered to be representative of the total corpus mucosa, and were averaged to determine the percentage that each component contributed to the mucosa of the gastric body. The corpus mucosal weight was calculated from the weight of the corpus and percentage of the wall that was mucosa. The mucosa of the gastric corpus as a fraction of the animal ranged from 1.1 g/kg for man to 4.3 for bullfrog, relative to body weight; or 12 g/m^2 for rat from a previous study to 62 for dog, relative to body surface area. The combined weight of mammalian parietal and chief cells was a fairly constant 53-58% of the corpus mucosa In the corpus mucosa of the guinea pig, there was a uniform distribution of parietal and chief cells. The parietal cell weight was 5 x the weight of the chief cells. In man, dog and cat, the parietal cell weight fraction increased toward the antrum, while that of chief cells decreased. Using data collected from the literature, "maximal" acid secretory rates were correlated with the total parietal cell weight and number of parietal cells. The maximal secretory rate expressed as mEq/hr/g of parietal cell varied among 5 mammals (man, dog, cat, guinea pig, and rat) by a factor of 5.3. Mean parietal cell volumes calculated from the weight of parietal cells divided by number of parietal cells ("parietal cell mass") are several times larger than mean volumes estimated from raw mean cell diameters. For interspecies comparison, expressing total parietal cell weight or maximal secretory rate relative to body surface area is not demonstrably better than normalizing these variables with respect to body weight.

0806 DIFFERENTIAL LOCALIZATION OF CELL SURFACE AND SECRETORY COMPONENTS IN RAT INTESTINAL EPITHELIUM BY USE OF LECTINS. *(E.)* Etzler, M. E.; Branstrator, M. L. (Dep. Biochem. Biophys., Univ. California, Davis). *J. Cell Biol.* 62(2):329-343, 1974.

Sections through various levels of small intestine from adult male Sprague-Dawley rats were examined by fluorescence microscopy after treatment with fluorescein isothiocyanate-labeled lectins from *Dolichos biflorus*, *Lotus tetragonolobus*, *Ricinus communis*, and *Triticum vulgare* (wheat germ). The animals were sacrificed by decapitation, and segments of the intestine removed next to the pylorus, and at 2-cm, 5-cm, 10-cm, 20-cm, and 25-cm distances from the pylorus. In some experiments, segments were also removed at the 50- and 75-cm levels, as well as 5-cm proximal to the cecum. The sections were mounted on slides and treated with approximately 100 μl of the appropriate fluorescein isothiocyanate-conjugated lectin at concentrations of 0.5-2 mg/ml. *L. tetragonolobus*, *R. communis*, and *T. vulgare* lectins reacted with the microvillar portion of the epithelial cells lining the crypts and villi in sections of intestine adjacent to the pylorus. This pattern of reactivity was sharply altered along the first 15 cm of intestine so that in sections distal to this point the luminal surfaces of only those epithelial cells in the crypts and at the base of the villi reacted with the *L. tetragonolobus* and *R. communis* lectins, whereas the wheat germ lectin reacted with the surfaces of the cells lining the villi. In sections from the distal end of the small intestine, all three lectins reacted with the surfaces of cells only at the base of the villi and in the crypts. The findings suggest that: 1) throughout most of the adult rat small intestine changes occur in the carbohydrate portion of the microvillar surface of the epithelial cells as these cells differentiate and move up the villi; 2) these changes in cell surface are related to the region of the intestine in which the villi are located; and 3) the carbohydrate nature of the secretory material of the goblet cells varies in different regions of the small intestine.

0807 STUDIES OF INTESTINAL ABSORPTION UNDER PATHOLOGICAL CONDITIONS. I. LIGHT MICROSCOPIC STUDIES OF INTESTINAL DAMAGE CAUSED BY SODIUM LAURYL SULFATE. *(E.)* Sugimura, F. (Nihon Univ. Sch. Med., Tokyo, Japan). *Nihon Univ. J. Med.* 16(1):25-37, 1974.

The effects of normal saline, 50% glucose, and 5% sodium lauryl sulfate (SLS) on the intestine were studied by light microscopy using *in vivo* preparations of unanesthetized adult male rabbit proximal jejunum. A 3 cm segment of small intestine was ligated on both ends and removed distal to a point 20 cm from the pylorus; these segments served as histological controls. The intestine was then ligated at a point 15 cm from the previous distal ligation, and either 10 ml of normal saline, 10 ml of 50% glucose solution, or 10 ml of 5% SLS solution was injected by syringe in this 15 cm segment of the intestine. After allowing the solution to remain in the loop of the intestine for 5, 10, 15, 30, and 60 min, a 3 cm segment was successively removed after each time period and examined microscopically. The epithelium was well

preserved 60 min after instillation of normal saline and nearly physiologic. Except for marked eosinophilic changes with the 50% glucose solution, only mild increases in goblet cells and very mild desquamation of the epithelium were noted; the striated border remained intact. The SLS solution caused desquamation of the epithelium, disappearance of the striated border, and spreading lytic degenerative changes which started on the surface of the villi and then extended to the entire villi and to the muscularis propria. In the area of focal degeneration, disappearance of the epithelial cells, indistinctiveness of the cell borders, prominent nuclei, and localized necrotic changes were noted. It is concluded that SLS has a toxic effect on the intestine. The initially focal and then spreading lytic changes of the villi and the muscularis propria appear to be due to permeation of the surface-active agent and demonstration of the proteins by SLS. Disappearance of the striated borders and obscuring of the cell borders and the nuclei are probably due to dissolution of biological membranes by SLS.

0808 STUDIES OF INTESTINAL ABSORPTION UNDER PATHOLOGICAL CONDITIONS. II. ELECTRON MICROSCOPIC STUDIES OF INTESTINAL DAMAGE CAUSED BY SODIUM LAURYL SULFATE. *(E.)* Sugimura, F. (Nihon Univ. Sch. Med., Tokyo, Japan). *Nihon Univ. J. Med.* 16(1):39-50, 1974.

In vivo effects of 5% sodium lauryl sulfate (SLS) on the intestinal epithelium in unanesthetized adult male rabbits were studied by electron microscopy. A 3 cm segment of jejunum was excised beginning from a point 20 cm from the pylorus to serve as a control. The intestine was then ligated at a point 15 cm from the previous ligation and into this 15 cm segment of jejunal tube 10 ml of 5% SLS was injected. After 5, 10, 15, 30, and 60 min, 3 cm segments of the intestine were successively ligated and excised, and examined by scanning electron microscopy or electron microscopy. Sixty min after the instillation of 5% SLS, a scanning electron microscopic view showed a marked reduction in the height of small intestinal villi, poorly defined individual villus, and what appeared to be a viscous substance covering the surface of the villi. It is suggested that this substance is a mixture or a conjugate of SLS with degenerated fuzzy coat, degenerated substance from the damaged portion of the intestine, and the secretions from the intestinal glandular cells. Under electron microscopy, the microvilli showed an altered arrangement, decreased height, and aggregation or disappearance after instillation of SLS. The formation of a terminal web was indistinct. The mitochondria and the majority of the lysosomes showed degeneration or disappearance. Some endoplasmic reticula showed saccular dilatation of the inner lumen, and the Golgi apparatus showed marked dilatation of Golgi vesicles. The intercellular spaces were widened and accumulation of absorbed substances was seen. Alterations in the ultramicroscopic structures of absorptive cells were more marked in those organelles which are involved in protein metabolism, leading to the conclusion that the tissue-damaging effect of SLS is basically due to its protein denaturing properties. These findings suggest that SLS causes an abnormality in intestinal absorption and is suitable to produce a pathologic intestinal model for the study of intestinal absorption in a pathological state.

0809 THE BURSA OF FABRICIUS: A CENTRAL ORGAN PROVIDING FOR CONTACT BETWEEN THE LYMPHOID SYSTEM AND INTESTINAL CONTENT. *(E.)* Schaffner, T.; Mueller, J.; Hess, M. W.; Cottier, H.; Sordat, B.; Ropke, C. (Dep. Pathol., Univ. Bern, Switzerland). *Cell Immunol.* 13(2):304-312, 1974.

The functions of the avian bursa of Fabricius were examined in male White Leghorn chickens. Fifteen chickens were fed 1.0 ml of colloidal carbon 3 times/day for 1 week and were sacrificed 10 days after the last feeding. Only the bursal tufts and follicles contained carbon particles. There was no appreciable carbon deposition in the epithelium outside the tufts or in the cortical zone. Colloidal carbon (0.1 ml) was administered *via* cloacal irritation to a total of 30 birds and groups of 5 animals sacrificed at various intervals from 15 min to 16 hr later. Within 15 min after carbon administration by this route, particles were present in the epithelial tufts; at 2 hr, they appeared in subepithelial locations; and at 16 hr, carbon aggregates were observed throughout the medullary portion of bursal follicles. The uptake of particles of different quality and size was studied following topical administration of 0.1 ml of colloidal thorium dioxide (thorotrast) or of a suspension containing latex spheres of different sizes. Twenty-four hr after administration, latex particles from 0.09 to at least 1.1 μm diameter and thorotrast particles also entered the bursal tissue, and were seen in the epithelial tufts, in macrophages and/or between lymphoid cells of the follicles. Bacteria (*Escherichia coli* or *Staphylococcus albus*) were administered in a suspension (0.1 ml) containing 5×10^9 microorganisms/ml directly into the bursal duct. No entry of living bacteria into the lymphoid tissue was observed by light or electron microscopy. The epithelial tuft cells which transported the inert particles administered in these studies are distinctively equipped with lytic enzymes. Based on these findings, it seems reasonable to assume that particulate material of microbial origin, would, upon penetration, be degraded rapidly and efficiently by cells of the epithelial tufts. Such a protective mechanism may yield antigenic fragments having an immunogenic role in the bursa or elsewhere.

0810 RECOVERY OF FUNCTION AND STRUCTURE OF DOG ILEUM AND COLON FOLLOWING TWO HOURS' ACUTE ISCHAEMIA. *(E.)* Robinson, J. W. L.; Haroud, M.; Winistorfer, B.; Mirkovitch, V. (Cantonal Univ. Hosp., Lausanne, Switzerland). *Eur. J. Clin. Invest.* 4(6): 443-452, 1974.

Loops of mongrel dog small intestine and colon were subjected to 2 hr acute ischemia under pentobarbital anesthesia (30 mg/kg) and their recovery studied simultaneously by functional and histological examination. The dogs were reoperated 1, 3, or 7 days after the 2-hr total ischemia for excision and exam-

ination of the treated loops and an adjacent control. Immediately after the ischemia, both organs showed serious necrotic lesions of the mucosal surface, their active transport capacities were abolished, mucosal Na^+-K^+-ATPase activities were reduced, and a significant liberation of acid phosphatase into the efferent blood was observed. The lesions of the ileal loops were more severe than those of the colonic loops. The ileal loops had a finite, but greatly reduced function 24 hr after the trauma. After 3 days, amino-acid transport had normalized but sugar transport was still reduced. Histological examination showed that epithelial cells covered the villi of most samples after 24 hr, and that normalization was almost complete after 3 days. All parameters were normal after 1 week. In 5 of 8 loops, the colonic mucosa was replaced by a compact polymorphonuclear infiltration bathing in a hemorrhagic exudate 24 hr after the ischemic period; it was evidently quite functionless. In 3 loops the mucosa was preserved, but only 1 had functional activity. Three days after the trauma, functional activity was finite, but lower than that of the control loops; histological examination revealed a disorganized mucosa, often with ulcers surrounded by relatively healthy tissue. At 7 days, the mean transport capacity was normal but there were still pathological signs at the microscopic level. The findings indicate that the ileum is more sensitive to ischemic damage, but has a greater regenerative potential than the colon. There was a good correlation between the functional and structural state of the tissues.

0811 ULTRASTRUCTURAL EFFECTS OF DIETARY GALACTOFLAVIN ON MOUSE HEPATOCYTES. *(E.)* Tandler, B.; Hoppel, C. L. (Sch. Med., Case Western Reserve Univ., Cleveland, Ohio). *Exp. Mol. Pathol.* 21(1):88-101, 1974.

The ultrastructural effects of a galactoflavin-supplemented (60 mg/kg of diet), riboflavin-free diet were chronologically examined in mouse hepatocytes to compare the effects of this drug with those produced by simple riboflavin deficiency. Control animals were maintained on a complete diet containing riboflavin and lacking galactoflavin. Animals were sacrificed by decapitation every 1-3 days from day 3-22, and on day 28 after initiation of the experimental diet. Some animals with signs of severe deficiency at day 20 or 21 were placed on a normal diet and given a daily i.p. injection of 0.35 mg riboflavin. Recovering animals were sacrificed daily until day 5, 7 and on day 10 after beginning the normal dietary program. Liver specimens were studied by electron and light microscopy. In animals on the experimental diet, the mitochondrial population suffered the greatest changes. Beginning with day 3, the mitochondria first became elongated, then later rounded up to become somewhat enlarged spheres. By day 20-22 the mitochondria reached diameters of about 6 μm, and had cristae that were largely confined to the periphery of the organelles. Throughout the course of enlargement, spontaneously partitioned mitochondria of all sizes were common. When the mice with advanced ariboflavinosis were injected with riboflavin, mitochondrial recovery took place over a period of more than 10 days. Division of the enlarged mitochondria proceeded by medial attenuation, rather than by partition formation and constriction. The mechanism that selects which of the two modalities of mitochondrial replication will prevail remains undetermined.

0812 MESENCHYME SPECIFICITY IN RODENT SALIVARY GLAND DEVELOPMENT: THE RESPONSE OF SALIVARY EPITHELIUM TO LUNG MESENCHYME IN VITRO. *(E.)* Lawson, K. A. (Hubrecht Lab., Utrecht, Netherlands). *J. Embryol. Exp. Morphol.* 32(2):469-493, 1974.

0813 PROXIMAL LIMITS OF THE GASTRIC ANTRUM. *(It.)* Brizzi, E.,; Serantoni, C.; Ciani, P. A.; Marinelli, R.; Fulignati, A. (Dep. Topogr. Anat., Univ. Florence, Italy). *Chir. Gastroenterol.* 8(2):137-152, 1974.

0814 THE GASTROEPIPLOIC NERVE IN MAN. *(It.)* Brizzi, E.; Serantoni, C.; Ciani, P. A.; Fulignati, A.; Marinelli, R. (Dep. Topogr. Anat., Univ. Florence, Italy). *Chir. Gastroenterol.* 8(1): 3-16, 1974.

0815 THE ANATOMY OF THE SUPERIOR MESENTERIC PLEXUS AND NERVES OF THE SMALL INTESTINE IN MAN. *(Rus.)* Sharov, V. A. (Med. Stomatol. Inst., Moscow, USSR). *Arkh. Anat. Gistol. Embriol.* 67(9): 106-110, 1974.

0816 THE PERMEABILITY PATHWAYS IN THE WALLS OF INTESTINAL SUBMUCOSAL ARTERIOLES IN ACUTE ANGIOTENSIN-INDUCED HYPERTENSION IN RATS. *(E.)* Thorball, N.; Olsen, F. (Dep. Anat. C, Univ. Copenhagen, Denmark). *Acta Pathol. Microbiol. Scand* [*A*] 82(6):683-689, 1974.

0817 FINDING OF GANGLION CELLS IN THE MUCOSA OF THE COLON, SIGMOID, AND RECTUM. *(Ger.)* Lassmann, G. (Neurol. Inst., Univ. Vienna, Austria). *Virchows Arch.* [*Pathol. Anat.*] 365(3):257-261, 1975.

0818 FUNCTIONAL MORPHOLOGY OF THE PANCREAS ASSOCIATED WITH VARIOUS DIETS. *(Rus.)* Permiakov, N. K.; Titova, G. P.; Podolskii, A. E. (N. V. Sklifosovskii Res. Inst. Emerg. Med. Treat., Moscow, USSR). *Arkh. Patol.* 36(10):25-33, 1974.

0819 INVESTIGATION OF LOCALIZATION OF MONOAMINE OXIDASE (EC 1.4.3.4.) IN SUBFRACTION OF RAT LIVER MITOCHONDRIA. *(E.)* Turska, E.; Turski, W. (Inst. Physiol. Biochem., Med. Acad., Lodz, Poland). *Enzyme* 19(4):140-148, 1975.

MORPHOLOGY, CELLULAR STRUCTURE AND HISTOCHEMISTRY

0820 BIOSYNTHESIS OF SERUM ALBUMIN IN RAT LIVER. ISOLATION AND PROBABLE STRUCTURE OF 'PRO-ALBUMIN' FROM RAT LIVER. *(E.)* Quinn, P. S.; Gamble, M.; Judah, J. D. (Univ. Coll. Hosp. Med. Sch., London, England). *Biochem. J.* 146(2):389-393, 1975.

See also, 0840, 0880, 0926, 0939, 0940, 0963, 1015, 1060, 1061, 1063, 1064, 1080, 1083, 1084, 1094, 1129, 1527.

ABSORPTION—EXCRETION—TRANSPORT

0821 EFFECT OF FORCE-FEEDING A VALINE-FREE DIET ON GASTROINTESTINAL FUNCTION OF RATS. *(E.)* Kimura, T.; Suzuki, S.; Yoshida, A. (Dep. Agric. Chem., Nagoya Univ., Japan). *J. Nutr.* 105(2):257-265, 1974.

The effect of an essential amino acid deficiency on gastrointestinal functions was studied by force-feeding male Wistar rats a 10% amino acid diet devoid of valine for several days, and then giving them a complete amino acid diet. The diets were force-fed 3 times daily at 4-hr intervals in amounts of 3.5, 3, and 3.5 g. Force-feeding of the valine-free diet for 4 or 11 days delayed the disappearance of dietary nitrogen and trichloroacetic acid (TCA)-soluble nitrogen from the gastrointestinal tract. A single feeding of the complete amino acid diet containing ^{14}C-labeled amino acids or [^{14}C]glucose to rats previously fed the valine-free diet, did not improve disappearance of the nitrogen. The absorption of dietary carbohydrate (4.25 μCi of [U-^{14}C]glucose) as well as the dietary nitrogen was delayed after feeding the valine-free diet, but the delayed absorption of the dietary nitrogen was improved after 1 day of feeding the complete amino acid diet to rats previously fed the valine-free diet. In spite of the delayed disappearance of dietary ^{14}C-labeled amino acids or [^{14}C]-glucose from the gastrointestinal tract, the incorporation of these dietary ^{14}C-labeled amino acids into liver lipids was not different in rats fed the complete amino acid diet and in those fed the valine-free diet. It is concluded that changes in disaccharidase activities in the intestinal mucosa were in parallel with changes in the disappearance of nutrients from the gastrointestinal tract under the experimental conditions used.

0822 A MODEL FOR PASSIVE TRANSPORT OF POTASSIUM BY THE STOMACH: EVIDENCE FROM IN VITRO STUDIES. *(E.)* Makhlouf, G. M. (Med. Coll. Virginia, Richmond). *Am. J. Physiol.* 227(6):1285-1288, 1974.

The mechanism of K^+ secretion by the stomach was studied in frog mucosal sacs bathed on the serosal side only with buffered Ringer solution containing 1-4 mM K^+. Microsamples of secretion were obtained and analyzed for H^+, Na^+, and K^+ by flame photometry. The potential difference was measured continuously with K^+-free saline (0.1 M NaCl) agar bridges. Secretory K^+ concentration was linearly related to serosal K^+ concentration. Concentrations ranged from 3.5 mM at 1 mM serosal K^+ to 11.9 mM at 4 mM serosal K^+. The slope of the relation indicated that K^+ was concentrated 3-fold in secretion. The initial potential difference (39.7 mV) fell with the start of secretion. The extent of the fall corresponded to the expected liquid junction potential (E_j) between the acid contents and the saline agar bridge. The potential difference during secretion, corrected for E_j, was similar to the initial presecretory potential difference (38.5) and somewhat higher than the mean K^+ equilibrium potential. Given the high K^+ permeability of the mucosa, the electrical gradient was of sufficient magnitude to determine a 3-fold concentration gradient in the water of secretion. The latter represented the common but independent link of H^+ and K^+ secretion, thus providing a ready explanation for the correlation of H^+ and K^+ flux rates and of H^+ and K^+ concentrations. It was not determined whether the pathway for the movement of K^+ into the lumen was intercellular or transcellular *via* a labile intracellular compartment. Either pathway would allow K^+ to be distributed at or close to electrochemical equilibrium in a fluid osmotically determined by the active transport of H^+ and Cl^-.

0823 ALTERATION OF THE PHYSICAL PATHWAYS THROUGH THE GASTRIC MUCOSA BY SODIUM TAUROCHOLATE. *(E.)* Birkett, D.; Silen, W. (Harvard Med. Sch., Boston, Mass.). *Gastroenterology* 67(6):1131-1138, 1974.

The effect of sodium taurocholate on gastric mucosal permeability was investigated using an *in vitro* preparation of rabbit fundic gastric mucosa mounted in an Ussing chamber and perfused with a physiological solution at 37 C. The permeability was measured by the mucosal to serosal flux of the water-soluble, non-lipid soluble, nonelectrolyte, [^{14}C]erythritol, [^{14}C]-mannitol, and [^{14}C]lactose. The transmucosal flux measurements were made during the 2nd and 3rd hr after mounting in the Ussing chamber by adding 5 μCi of the nonelectrolyte to the mucosal solution and taking 1000 μl samples from the serosal solution for scintillation counting at 15-min intervals. The flux rate of erythritol in normal tissue was 1.85 pM/cm^2/sec and was increased by 20 mM sodium taurocholate in the mucosal solution at pH 7.0 and pH 3.0 to 5.46, and 25.48 pM/cm^2/sec, resp. after 1 hr, demonstrating a pH-dependent effect. At pH 3.0, sodium taurocholate in the mucosal solution at doses of 2.5, 5, and 10 mM increased the mucosal to serosal flux rate to 3.98, 5.81, and 13.52 pM/cm^2/sec, resp., after 1 hr, demonstrating a dose-related effect. There was no statistical difference between the mucosal to serosal flux of erythritol with 20 mM sodium taurocholate in the mucosal or serosal solutions. The fluxes of mannitol and lactose were altered in a similar direction by sodium taurocholate, but the absolute flux of these substances was less than that of erythritol because of the larger size of the molecule. It is

suggested that sodium taurocholate causes an increase in permeability of the gastric mucosa through water-filled channels, which may be responsible for the increased transmucosal flux of H^+ and Na^+ caused by bile salts.

0824 EFFECT OF SEVERE ANOXIA ON THE PERMEABILITY OF GASTRIC MUCOSA. *(E.)* Birkett, D.; Silen, W. (Harvard Med. Sch., Boston, Mass.). *Proc. Soc. Exp. Biol. Med.* 148(1):256-260, 1975.

The effect of severe anoxia on the H^+ back diffusion and the permeability of the gastric mucosa to erythritol was investigated *in vitro*, using gastric mucosa from male New Zealand white rabbits. Each side of the mucosa was perfused separately with Ringer's solution containing 136 mM NaCl, 5 mM KCl, 3.5 mM $CaCl_2 \cdot 2H_2O$, and 1.2 mM $MgCl_2 \cdot 6H_2O$, and aerated with either 100% O_2 for the control or 100% N_2 to produce severe anoxia. The mucosal and serosal jackets were maintained independently at pH 7.4. The H^+ concentration of the mucosal solution was altered, and the back diffusion determined from the amount of 0.05 N HCl needed to maintain constant pH. To determine erythritol flux, tissues were perfused with phosphate-buffered Ringer's solution (pH 7.0) containing 1 mM erythritol to which increments of ^{14}C erythritol were added. The H^+ back diffusion rate increased as the pH fell in the oxygenated tissues. The rate was greater in the unoxygenated tissues but to a significant degree only after 1.5 hr. Erythritol flux rate from mucosal to serosal solution rose after 45 min of anoxia but not to a statistically significant degree. The 100% N_2 caused an immediate fall to zero of the electric potential, while the controls showed a slow, gradual decline. It is concluded that anoxia increases mucosal permeability only when prolonged to an extent beyond that likely to be found *in vivo*.

0825 INFLUENCE OF POTASSIUM CHLORIDE ON ALCOHOL ABSORPTION. *(E.)* Redetzki, H. M.; Dees, W. L.; Maddox, J. H. (Louisiana State Univ. Sch. Med., Shreveport). *Life Sci.* 15(6):2145-2156, 1974.

The effects of potassium chloride on alcohol absorption was investigated in female Sprague-Dawley rats. Alcohol was administered either through a stomach tube intragastrically (4 g/kg) or i.v. (2 g/kg). Potassium chloride was added to the intragastrically administered alcohol solutions in quantities to provide dosages of 215 mg/kg, 430 mg/kg and 860 mg/kg. Plasma potassium levels were determined by flame photometry. Simultaneous intragastric administration of the 2 larger doses of KCl with ethanol significantly reduced blood alcohol levels and diminished manifestations of alcohol intoxication. It was shown with parenteral administration of alcohol that the effect was not related to an acceleration of alcohol metabolism. Equimolal concentrations of NaCl were unable to duplicate these effects, thus indicating that they are specific actions of the potassium ion. Analysis of alcohol concentrations of gastric and intestinal content as well as *in situ* studies with animals whose stomachs were ligated at the pylorus revealed that KCl interferes with the absorption of alcohol through inhibition of gastric absorption and gastric emptying.

0826 INFLUENCE OF GLUCAGON ON THE HUMAN JEJUNUM. *(E.)* Hicks, T.; Turnberg, L. A. (Manchester R. Infirm., England). *Gastroenterology* 67(6):1114-1118, 1974.

Jejunal net transport of salt and water was measured using a triple lumen tube perfusion technique in a group of 22 normal adult volunteers ranging in age from 18 to 34 yr. Commercially available porcine pancreatic glucagon given by infusion for 90 min reduced the absorption of Na^+, Cl^-, and water. A dose-response curve for glucagon indicated that the effect on ion transport was maximal to 0.6 µg/kg/hr, and this effect decreased as the dose was increased, until, at a dose of 2.7 µg/kg/hr, ion transport was unaffected. The mean transit time was increased by glucagon in a dose of 1.2 µg/kg/hr, and the calculated volume and diameter of the jejunum was also increased. The levels of plasma concentration calculated to have been achieved by these infusions were above those reported under physiological conditions, but they were within the range which could conceivably be found in pathological conditions where these observations may have some relevance.

0827 JEJUNAL PERFUSION OF SIMPLE AND CONJUGATED FOLATES IN MAN. *(E.)* Halsted, C. H.; Baugh, C. M.; Butterworth, Jr., C. E. (Johns Hopkins Univ. Sch. Med., Baltimore, Md.). *Gastroenterology* 68(2):261-269, 1975.

The assimilation of pteroylpolyglutamate was studied in 5 healthy adult volunteers using the jejunal perfusion technique. Two µM each of ^3H-labeled pteroylpolyglutamate and synthetic ^{14}C-labeled pteroylheptaglutamate/liter of isotonic saline were perfused and aspirated by multilumen tubes. Polyethylene glycol was also added as a non-absorbable marker. A turbidometric method was used to determine polyethylene glycol concentration, counting was done by liquid scintillation counter, and intestinal aspirates were also subjected to analytical ion exchange column chromatography. The folates were also exposed to intestinal juice *in vitro* under similar physical conditions as in the *in vivo* studies. In each subject the concentrations of both types of folate decreased progressively with intestinal passage: a rate of 14.94 and 10.51 nM/min for the ^3H- and ^{14}C-folates, resp. In the unabsorbed fraction, 16.6% remained as ^{14}C-pteroylheptaglutamate, while 30.8% was in the form of its hydrolytic derivatives. In contrast, 84-95% of the ^{14}C was in the form of the parent compound *in vitro*. It is concluded that contact of conjugated folate with the intestinal mucosa is required for its digestion, and that progressive hydrolysis of pteroylheptaglutamate may be an integral part of its absorptive mechanism.

0828 THEOPHYLLINE, CYCLIC AMP, CHOLERAGEN, AND ELECTROLYTE TRANSPORT BY RABBIT ILEUM. *(E.)* Powell, D. W.; Farris, R. K.; Carbonetto, S. T. (Univ. North Carolina Sch. Med., Chapel Hill). *Am. J. Physiol.* 227(6):1428-1435, 1974.

The effects of theophylline, cyclic AMP (cAMP), and purified cholera toxin (choleragen) on electrolyte

transport were studied in the *in vitro* rabbit ileum in Ringer solution. Theophylline (10 mM) and cAMP (7.5 mM) were added to the serosal bathing solution along with equimolar mannitol in the mucosal solution to balance the osmolality. Choleragen in concentrations of 2 μg/ml was added to the mucosal solution after mounting intestine in 10 mM mannitol-Ringer. Tissues were continuously short-circuited except for brief periods when the open-circuit potential difference was determined. Radioactive isotope (^{22}Na, ^{24}Na, ^{36}Cl) was added to the mucosal and/or serosal bath 10-15 min after mounting. Seven 20-min flux periods were measured beginning 15 min after the isotope was added in the theophylline and cAMP experiments; 4 15-min fluxes, beginning at 175 min, were measured in the choleragen studies. The effects of all 3 agents were basically the same. All 3 caused a short-lived stimulation of short-circuit current, a sustained increase in potential difference, a reduction in electrical conductance, and a secretion of Na^+ as well as Cl^-. The Na^+ and Cl^- secretion was inhibited by ouabain. Theophylline caused Cl^- secretion in HCO_3^--free solution and probably HCO_3^- secretion in Cl^--free solution. Theophylline had no effect on Na^+ transport in the absence of Cl^- and HCO_3^- and no effect on anion transport in the absence of Na^+. These effects are similar to those previously demonstrated with cholera toxin. This investigation lends support to the concept that cAMP is involved in the electrolyte secretion of cholera and suggests that increased cellular cAMP levels stimulate the neutral secretion of NaCl and/or $NaHCO_3$.

0829 INTESTINAL ABSORPTION OF SOME HETEROCYCLIC AND AROMATIC AMINO ACIDS FROM THE AGEING GUT. *(E.)* Penzes, L.; Boross, M. (Univ. Med., Budapest, Hungary). *Exp. Gerontol.* 9(5/6):253-258, 1974.

The influence of age on intestinal absorption of L-tryptophan, L-phenylalanine and L-proline was studied in 6-, 12-, and 27-month old female Wistar rats. The amino acids were applied in the following initial concentrations: tryptophan, 1, 2, 5, and 10 mM; phenylalanine, 5, 10, 20, and 30 mM; and proline, 10, 20, 40, and 80 mM. Absorption rates were calculated using the Lineweaver-Burk relationship to establish the Michaelis constants (K_m and V_{max}) for each amino acid. The young animals had the smallest numerical K_m values in each case: typtophan, 1.4, 6.8, and 3.1 for young, adult, and old rats, resp.; phenylalanine, 10.6, 17.7, and 42.4 mM, resp.; and proline, 23.3, 28.0, and 36.6 mM, resp. The maximal transport velocities (V_{max}), given in M/g dried intestine/20', were as follows: tryptophan, 75.7, 153.1, and 85.0 for young, adult, and old rats, resp.; phenylalanine, 221.1, 448.4, and 724.6, resp.; and proline 409.5, 445.2, and 879.5, resp. The observed K_m values indicate that these diminish with increasing lipophilic characteristics of the amino acid. The V_{max} was in inverse ratio to lipid solubility. This indicates that a high numerical value for K_M corresponds to a considerable value of V_{max}, which results, in general, in an intense rate of absorption. It is concluded that a high numerical value for K_m corresponds to a considerable value of V_{max}, which results, in general, in an intense rate of absorption. It is concluded age-dependent phenomenon.

0830 EFFECT OF PHENOBARBITAL ON INTESTINAL CHOLIC ACID ABSORPTION IN THE RAT. *(E.)* Ostrower, V. S.; Coan, P.; Kern, Jr., F. (Univ. Colorado Med. Cent., Denver). *Gastroenterology* 67(6):1162-1168, 1974.

Kinetic data on the passive jejunal and active ileal transport of cholic acid were determined in control and phenobarbital-treated female Sprague-Dawley rats using an *in vivo* isolated loop perfusion technique. The treated rats received s.c. sodium phenobarbital, 80 mg/kg/day for 8 days. Perfusion solutions for individual experiments were labeled with [^{14}C]cholic acid, brought to final volume with Krebs-bicarbonate, and to a final pH of 8; final concentrations varied between 0.1 and 10 mM. Perfusions were carried out at a calibrated rate of 0.52 ml/min for 60-120 min. During perfusions, consecutive 5-min aliquots of bile were collected directly into counting vials and the radioactivity in bile and perfusion solutions counted in a liquid scintillation spectrometer. Transport in the jejunum was consistent with a passive transport mechanism. The passive diffusion coefficient for jejunal transport for control animals (136 pM/min/cm/100 g/mM) was not significantly different than that for treated animals (147 pM/min/cm/100 g/mM). The maximum absorption rate for active ileal transport in phenobarbital-treated rats (5300 pM/min/cm/100 g) was twice that for control animals (2239 pM/min/cm/100 g). This suggests that the active bile salt ileal transport mechanism can be enhanced by phenobarbital. This effect may play a role in phenobarbital-induced alterations of the enterohepatic circulation of bile salts and may be of benefit in some bile salt malabsorption syndromes.

0831 UPTAKE OF CALCIUM BY MICROVILLOUS MEMBRANES OF INTESTINAL EPITHELIAL CELLS. *(E.)* Hudson, D. A. (Dunn Nutr. Lab., Univ. Cambridge, England). *Life Sci.* 16(1):117-129, 1975.

Some of the characteristics of uptake of Ca^{++} by microvillous membranes from the small intestine of male and female Dutch rabbits were investigated in the absence and presence of ATP. Uptake of Ca^{++} was measured by a Millipore filtration technique. The membranes (100 μg of protein) were incubated at 37 C for 20 min in a medium containing 10 mM Tris Hepes (pH 7.4), 0.2 μCi ^{45}Ca, and $CaCl_2$ to the desired final concentration. In the absence of ATP, Ca^{++} uptake was independent of temperature (3-37 C), reduced by the presence of other cations, and dependent upon pH and the number and type of "binding" sites. At all Ca^{++} concentrations tested up to 5×10^{-3} M, addition of ATP (0.1-10 mM) at 37 C caused a reduction in uptake relative to controls. Mg^{++} (2 mM) caused a partial reversal of this ATP-induced reduction. Between 5 and 10 mM Ca^{++}, addition of ATP increased the amounts of the ion apparently associated with the membranes. The type, extent, and stability of the association of Ca^{++} with microvillous membranes is probably determined by the number and type of dissociable anions capable of complex formation. Two non-specific factors may have contributed to the observed relationship between Ca^{++} concentration and its uptake in the presence of ATP: 1) the chelating properties of the nucleotide and its breakdown products; and 2) an increase in the ionic strength of the incubation medium. It is suggested that stimulation of

Ca^{++} uptake by ATP when the ion was present at concentrations of 5 mM and higher may be explained without invoking any causal relation between nucleotide hydrolysis and ion movement.

0832 THE SITE OF ZINC ABSORPTION IN THE RAT SMALL INTESTINE. *(E.)* Emes, J. H.; Arthur, D. (Dep. Nutr., Univ. Guelph, Canada). *Proc. Soc. Exp. Biol. Med.* 148(1):86-88, 1975.

The site of zinc absorption was determined by comparing the zinc distribution patterns in the small intestine of zinc-deficient rats with controls, and by measuring ^{65}Zn absorption. To study distribution, the small intestines were cut into 5 equal segments, wet-ashed, and measured for zinc content by atomic absorption spectrometry. ^{65}Zn absorption was measured by gamma well detector after incubation of intestine with $^{65}ZnCl_2$. Zinc distribution was highest in the 5th segment and lowest in the 2nd in controls, while deficient rats showed a constant small but significant increase from the 2nd segment to the distal ileum, though total zinc concentration was reduced. ^{65}Zn absorption showed a steady increase from duodenum to distal ileum on a per weight basis, while on a per unit length basis showed a bimodal pattern of uptake. It is suggested zinc absorption occurs most actively in the distal ileum.

0833 THE TRANSMISSION OF ^{125}I-LABELED IMMUNOGLOBULIN G BY PROXIMAL AND DISTAL REGIONS OF THE SMALL INTESTINE OF 16-DAY-OLD RATS. *(E.)* Morris, B. (Dep. Zool., Univ. Nottingham, England). *J. Physiol. (Lond.)* 245(1):249-259, 1975.

Labeled immunoglobulin transmission from the small intestine of 16 day Wistar rats was studied to determine the region most important in its absorption. Histological studies were also done to determine the extent of distal intestine lined by epithelial cells with large supranuclear vacuoles. Rat IgG was labeled with ^{125}I and 2 ml injected into the proximal or the distal small intestine by incision through the body wall. The intestine was ligated proximal to the injection site. Blood was collected from the heart, and assayed for radioactivity, the protein content removed by centrifugation, and the radioactivity determined for both precipitate and supernatant. Values were expressed as concentration quotients (cpm/0.1 ml serum protein to cpm/0.1 ml of injected preparation). Sections of intestinal wall were fixed in Bouin's fluid and stained in either Ehrlich's hematoxylin and eosin, or Harris's hematoxylin and alcian blue. The concentration quotients from 2 and 3 hr after injection into the proximal intestine were high (0.0130–0.1031) while those from an equal time after injection into the distal portion were very low (0.00062–0.0096). The histological studies demonstrated that the extent of the terminal gut region increased with age and was characterized by the new type epithelial cells with large supranuclear vacuoles. It is concluded that a greater amount of IgG absorption occurs in the proximal intestine due to cell type changes in the distal regions.

0834 COMPARISON OF TRANSPORT MECHANISMS IN ISOLATED ASCENDING AND DESCENDING RAT COLON. *(E.)* Yau, W. M.; Makhlouf, G. M. (Med. Coll. Virginia, Richmond). *Am. J. Physiol.* 228(1):191-195, 1975.

The transport properties of the ascending and descending colon of 19 unfasted male albino rats were compared *in vitro*. The mucosal tubes were everted and cut into equal proximal and distal halves. Each half was ligated at one end and tied to a cannula at the other. They were filled with and immersed in a bathing solution containing (in mM/liter) 145.6 Na^+, 5.3 K^+, 1.3 Ca^{++}, 1 Mg^{++}, 125.7 Cl^-, 24.2 HCO_3^-, 2 H_2PO_4, and 15 glucose (298.7 mOsm/kg). Water flux was measured gravimetrically and used to calculate net ionic flux. Potential differences were measured with agar bridges in the mucosal and serosal compartments. Na^+ and K^+ were measured by flame photometer. The net volume, anion, and total solute fluxes were similar in both colon segments, but their ionic components were not. The descending colon absorbed significantly less Na^+ and more HCO_3^-, as well as significant amounts of K^+. The ascending colon neither absorbed nor secreted K^+. In the serosal compartment, Na^+ and Cl^- concentrations increased, and HCO_3^- decreased, to different extents in the 2 segments. Most pronounced was the difference in the K^+ concentration, which decreased by about 26% in the ascending segment, but increased by about 36% in the descending segment. No consistent differences between the 2 segment types were found in the electrical gradient. In both, Na^+ absorption occurred against electrical and chemical gradients. It is concluded that a quantitative difference in Na^+ absorption, HCO_3^- secretion, and a qualitative difference in K^+ transport exists between the ascending and descending colon.

0835 NON-PARALLEL TRANSPORT OF MEMBRANE PROTEINS AND CONTENT PROTEINS DURING ASSEMBLY OF THE SECRETORY GRANULE IN RAT PAROTID GLAND. *(E.)* Wallach, D.; Kirshner, N.; Schramm, M. (Dep. Biol. Chem., Heb. Univ. Jerusalem, Israel). *Biochim. Biophys. Acta.* 375(1):87-105, 1974.

0836 TRANSPORT AND ELECTRICAL PHENOMENA IN RESTING AND SECRETING PIGLET GASTRIC MUCOSA. *(E.)* Forte, J. G.; Machen, T. E. (Inst. Anim. Physiol., Cambridge, England). *J. Physiol. (Lond.)* 244 (1):33-51, 1975.

0837 DISPOSITION OF N,N-BIS(PHENYLCARBAMOYLMETHYL)DIMETHYL AMMONIUM CHLORIDE IN THE RAT: AN INTERESTING EXAMPLE OF FIRST-PASS METABOLISM. *(E.)* Gibaldi, M.; Axelson, J. E.; Conway, W. D. (Sch. Pharm., State Univ. New York Buffalo). *J. Pharm. Sci.* 64(2):359-360, 1975.

0838 EFFECT OF ETHANOL ON THEOPHYLLINE ABSORPTION IN HUMANS. *(E.)* Koysooko, R.; Ellis, E. F.; Levy, G. (Sch. Pharm., State Univ. New York Buffalo). *J. Pharm. Sci.* 64(2):299-301, 1975.

0839 INTESTINAL CONDUCTANCE AND PERMSELECTIVITY CHANGES WITH THEOPHYLLINE AND CHOLERAGEN. *(E.)* Powell, D. W. (Univ. North Carolina Sch. Med., Chapel Hill). *Am. J. Physiol.* 227(6):1436-1442, 1974.

ABSORPTION–EXCRETION–TRANSPORT

0840 THE EFFECT OF PREGNANCY AND LACTATION ON FOOD INTAKE, GASTROINTESTINAL ANATOMY AND THE ABSORPTIVE CAPACITY OF THE SMALL INTESTINE IN THE ALBINO RAT. *(E.)* Cripps, A. W.; Williams, V. J. (Dep. Physiol., Univ. New England, Armidale, Australia). *Br. J. Nutr.* 33(1):17-32, 1975.

0841 INFLUENCE OF THE STATE OF DIETARY PROTEIN ON THE RATE OF AMINO ACID DIGESTION AND ABSORPTION IN THE SMALL INTESTINE OF RATS. *(Rus.)* Usacheva, N. T.; Nikolskaia, G. V.; Chernikov, M. P. (Inst. Nutr., Moscow, USSR). *Vopr. Pitan.* (6): 22-26, 1974.

0842 IMMUNOLOGIC CONTROL OF SOLUBLE PROTEIN ABSORPTION FROM THE SMALL INTESTINE: A GUT-SURFACE PHENOMENON. *(E.)* Walder, W. A.; Isselbacher, K. J.; Bloch, K. J. (Massachusetts Gen. Hosp., Boston). *Am. J. Clin.* 27(12):1434-1440, 1974.

0843 THE ABSORPTION OF ALG FROM THE GUT AND ITS LYMPHOCYTOPENIC EFFECT IN DOGS. *(E.)* Seifert, J.; Ring, J.; Brendel, W. (Surg. Clin., Univ. Munich, Germany). *Eur. Surg. Res.* 6(1):44-45, 1974.

0844 INTESTINAL ABSORPTION OF GLYCINE, L-ALANINE AND L-LEUCINE IN THE OLD RAT. *(E.)* Penzes, L. (Univ. Med., Budapest, Hungary). *Exp. Gerontol.* 9(5/6):245-252, 1974.

0845 THE INTESTINAL ABSORPTION OF VITAMIN B_{12}. *(E.)* Schjonsby, H.; Andersen, K. J. (Med. Dep. A, Univ. Bergen, Norway). *Scand. J. Gastroenterol.* 9(29):7-11, 1974.

0846 THE ACCUMULATION OF ADENOSINE IN RABBIT INTESTINAL MUSCLE. *(E.)* Hulme, M. E.; Weston, A. H. (Dep. Pharmacol., Univ. Manchester, England). *Br. J. Pharmacol.* 50(4):569-574, 1974.

0847 THE EFFECT OF PROTEIN SYNTHESIS INHIBITORS ON ZINC ABSORPTION BY RAT DUODENUM IN VIVO. *(E.)* Davies, N. T. (Rowett Res. Inst., Aberdeen, Scotland). *J. Physiol. (Lond.)* 244(1):38P-39P, 1975.

0848 THE EFFECTS OF ANGIOTENSIN ON FLUID TRANSPORT AND BLOOD FLOW IN RAT JEJUNUM. *(E.)* Bolton, J. E.; Munday, K. A.; Parsons, B. J. (Dep. Physiol., Univ. Southampton, England). *J. Physiol. (Lond.)* 244(1):27P-28P, 1975.

0849 EVALUATION OF CHROMIC OXIDE, GLYCEROL TRIETHER, AND β-SITOSTEROL AS FECAL FLOW MARKERS IN TWO SPECIES OF NONHUMAN PRIMATES. *(E.)* St. Clair, R. W.; Lehner, N. D. M.; Hamm, T. E. (Bowman Gray Sch. Med., Winston-Salem, N.C.). *Lipids* 10(1):25-32, 1975.

0850 REGULATION OF METABOLITE TRANSPORT IN RAT AND GUINEA PIG LIVER MITOCHONDRIA BY LONG CHAIN FATTY ACYL COENZYME A ESTERS. *(E.)* Shrago, E.; Shug, A.; Elson, C.; Spennetta, T.; Crosby, C. (Dep. Med., Univ. Wisconsin, Madison). *J. Biol. Chem.* 249(16):5269-5274, 1974.

0851 LIVER EXCRETORY MECHANISMS FOLLOWING PORTACAVAL SHUNT. *(E.)* Franco, D.; Chauvaud, S.; Grange, D.; Bismuth, H. (Paul-Brousse Hosp., Villejuif, France). *Eur. Surg. Res.* 6(Suppl. 1):2-3, 1974.

0852 BILIARY EXCRETION OF PHTHALEIN DYES: A SEPARATE PATHWAY FOR ROSE BENGAL? *(E.)* Mahu, J. L.; Arondel, J.; Dhumeaux, D.; Berthelot, P. (INSERM, Henri Mondor Hosp., Creteil, France). *Digestion* 10(4/5):366, 1974.

0853 THE MECHANISM OF AN INHIBITORY ACTION OF OXYTOCIN ON THE ISOTONIC FLUID TRANSPORT THROUGH THE GALL BLADDER EPITHELIUM. *(Rus.)* Iaremenko, M. S.; Kharlamova, O. N. (A. Bohomolets Inst. Physiol., Kiev, USSR). *Biull. Eksp. Biol. Med.* 77(11):10-12, 1974.

0854 ELECTRICAL PROPERTIES AND DIFFUSION POTENTIALS IN THE GALLBLADDER OF MAN, MONKEY, DOG, GOOSE AND RABBIT. *(E.)* Gelarden, R. T.; Rose, R. C. (Milton S. Hershey Med. Cent., Hershey, Pa.). *J. Membrane Biol.* 19(1/2):37-54, 1974.

See also, 0807, 0808, 0810, 1086, 1273, 1287.

MOTILITY

0855 FACTORS LIMITING GASTRIC EMPTYING DURING REST AND EXERCISE. *(E.)* Costill, D. L.; Saltin, B. (Dep. Physiol., Coll. Gymnast. Sports, Stockholm, Sweden). *J. Appl. Physiol.* 37(5):679-683, 1974.

A series of studies was undertaken to determine the effects of solute volume (200, 400, 600, 800 ml), temperature (5, 15, 25, 35 C), osmolarity (0-900 mOsm), and glucose concentration (139, 278, 556, 834 mM) on the rate of gastric emptying in 15 healthy

males who served as their own controls. Additional observations were made to assess the effects of exercise intensity on the rate at which 400 ml of a glucose (139 mM) solution was removed from the stomach. At rest the addition of even small amounts of glucose (> 139 mM) induced a marked reduction in the rate of gastric emptying. The volume of fluid remaining in the stomach 15 min after ingestion was somewhat greater when the solution was 35 C as opposed to colder fluids ($P < 0.05$). The rate of gastric emptying increased in proportion to the volume ingested, with a maximal rate of emptying attained at a volume of 600 ml. In general, exercise had no effect on gastric emptying until the working intensity exceeded 70% $V_{max}O_2$. These data demonstrate the importance of minimizing the glucose content of solutions ingested in order to obtain an optimal rate of fluid replacement. In combination with high-intensity exercise even small amounts of carbohydrate can block gastric emptying.

See also, 0826, 0870, 0875.

SECRETION AND METABOLISM

0856 THE PRESENCE OF CYTIDINE 5'-MONOPHOSPHOSIALIC ACID: GLYCOPROTEIN SIALYLTRANSFERASE IN HUMAN SALIVA. *(E.)* Nakamura, R.; Tsunemitsu, A. (Dep. Prev. Dent., Hiroshima Univ., Japan). *J. Dent. Res.* 54(1):188, 1975.

0857 CHEMISTRY OF GASTROINTESTINAL HORMONES. *(E.)* Wunsch, E. (Max Planck Inst. Biochem., Munich, Germany). *Helv. Med. Acta.* 37(5/6): 307-320, 1974.

0858 INACTIVATION OF GASTRIN BY LYSOSOMAL ENZYMES. *(Ger.)* Hunziker, H. R. (Canton. Hosp., Aarau, Switzerland). *Helv. Med. Acta* 37 (5/6):357, 1974.

0859 INTESTINAL GLUCAGON. *(Ger.)* Muller, W. A. (Massachusetts Gen. Hosp., Boston). *Helv. Med. Acta* 37(5/6):321-330, 1974.

See also, 0821.

SECRETION AND METABOLISM

Stomach

0860 EFFECT OF ANGIOTENSIN AND ACTH ON GASTRIC SECRETION. *(E.)* Nasca, J. D.; Johnson, C.; Nance, F. C. (Dep. Surg., Louisiana State Univ., New Orleans). *Am. Surg.* 40(12):709-712, 1974.

The effects of angiotensin (1.7 µg/min i.v.) and ACTH (40 U/24 hr i.m.) alone or in combination were studied on pentagastrin (2 µg/kg/hr i.v.)-stimulated gastric acid secretion in 4 fasted mongrel dogs with gastric fistulas. Baseline gastric acid output in pentagastrin-stimulated dogs was about 4-6 mEq H^+/15 min. Addition of a constant ACTH administration caused no significant change in the gastrin-stimulated secretion. With the addition of angiotensin to the ACTH, background acid output decreased an approximate 75%. Infusion of angiotensin alone into pentagastrin-stimulated dogs caused a 60% decrease in acid production. The inhibition of acid release by angiotensin is best explained by its ability to cause marked splanchnic vasoconstriction resulting in decreased blood flow which in turn has been shown to reduce gastric secretion. The potentiation of this effect by ACTH may be mediated by higher levels of circulating angiotensin caused by the stimulation of renin release by ACTH.

0861 PLASMA GASTRIN LEVELS AND GASTRIC ACID SECRETION IN DOGS DURING ADMINISTRATION OF URECHOLINE. *(E.)* Sjodin, L.; Nilsson, G. (Karolinska Inst., Stockholm, Sweden). *Scand. J. Gastroenterol.* 9(8):747-750, 1974.

The effects of bethanechol (Urecholine), a stable choline-ester, were studied on gastric acid secretion and plasma gastrin levels of 4 fasted dogs with Heidenhain pouches and gastric cannulas. Continuous i.v. infusion of bethanechol (50 µg/kg-hr) over a 4 hr period stimulated gastric acid secretion to a value 40% of the mean maximal rate induced by histamine (4 mg/hr). Gastric secretion was stimulated to 66% of the maximal histamine-induced rate by 100 µg/kg-hr bethanechol. Neither concentration of bethanechol, however, significantly altered serum gastrin levels. Both doses produced some cholinergic side effects including salivation and vomiting. These results indicate that parietal cells are more sensitive to stimulation by bethanechol than are gastrin-releasing cells. Possible explanations for this include: 1) basal serum gastrin levels are sufficient to induce acid secretion in the presence of direct stimulation by bethanechol; or 2) bethanechol is

stimulating release of substances which stimulate gastric acid secretion but which are not detected by the gastrin radioimmunoassay used in these studies.

0862 INTERACTION BETWEEN SECRETIN AND CHOLECYSTOKININ ON INHIBITION OF GASTRIC SECRETION IN MAN. *(E.)* Henriksen, F. W.; Jorgensen, S. P.; Moller, S. (Rigshosp., Copenhagen, Denmark). *Scand. J. Gastroenterol.* 9(8):735-740, 1974.

The inhibitory effects of secretin (0.5-1.0 U/kg/hr i.v.) and cholecystokinin (0.5 and 1.0 Ivy dog U/kg/hr i.v.), given alone or in combination, on pentagastrin (0.15, 1.0 or 6.0 µg/kg/hr i.v.)-induced gastric acid secretion were studied in 6 healthy male and 6 healthy female fasted volunteers. Gastric fluid secretion was significantly decreased (12%) only when subjects given 6.0 µg/kg/hr pentagastrin received 10 U/kg/hr secretin. Gastric acid secretion, however, was significantly inhibited by all inhibitory infusions except for the lowest doses of secretin and cholecystokinin alone. When the low doses of secretin and cholecystokinin were given in combination, however, gastric acid secretion was inhibited to an extent (28%) greater than the sum of the degrees of inhibition seen when secretin or cholecystokinin were given alone (14%). This was not true for the higher individual doses where the degrees of gastric acid inhibition were also significantly increased for both secretin alone (14-23%) and for cholecystokinin alone (14-15%). Since the lower doses of agents given in these studies are most likely within the range of those released endogenously, it is concluded that the inhibition of gastric acid secretion resulting from the interaction of secretin and cholecystokinin is of physiologic importance in man.

0863 THE EFFECT OF PENTAGASTRIN, EXOGENOUS SECRETIN, AND CHOLECYSTOKININ (CCK) ON THE INTRINSIC FACTOR (IF) SECRETION IN MAN. *(E.)* Vatn, M. H. (Ulleval Hosp., Oslo, Norway). *Scand. J. Gastroenterol.* [*Suppl.*] 9(29):53-56, 1974.

The effects of secretin and cholecystokinin on pentagastrin-stimulated intrinsic factor secretion were studied in fasted, healthy male and female volunteers. Three series of i.v. infusions were performed: 1) pentagastrin was given alone or in combination with secretin or cholecystokinin; 2) increasing doses of secretin or pentagastrin were given on the same day; 3) graded doses of pentagastrin were given on different days. Cholecystokinin caused a reduction of pentagastrin-stimulated intrinsic factor, acid, and pepsin output. Secretin caused a reduction of pentagastrin-stimulated acid and augmentation of pepsin output, whereas the output of intrinsic factor was unchanged. Basal intrinsic factor and acid output was reduced by increasing doses of secretin from 0.078 to 2.1 U/kg/hr, secondary to a reduction of volume. The output of pepsin was unchanged, owing to increased concentration. Stepwise increasing doses of pentagastrin, from 0.028 to 6.0 µg/kg/hr caused a continuous 2-hr secretion of intrinsic factor on a high plateau level, similar to that of pepsin. Infusion of a single dose caused a continuous low level secretion of intrinsic factor, contrasting the high level secretion for pepsin. The results suggest a dose-dependent relationship between pentagastrin and intrinsic factor, acid, or pepsin, with a different mode of secretion for the 3 substances of gastric juice.

0864 GASTRIC SECRETORY INHIBITION INDUCED BY THREE METHYL ANALOGS OF PROSTAGLANDIN E_2 ADMINISTERED INTRAGASTRICALLY TO MAN. *(E.)* Nylander, B.; Andersson, S. (Karolinska Inst., Stockholm, Sweden). *Scand. J. Gastroenterol.* 9(8):751-758, 1974.

The effects of 3 alkylated analogs of prostaglandin E_2 (PGE_2) (15(S)-15-methyl PGE_2 methyl ester, 16,16-dimethyl PGE_2 and 16,16-dimethyl PGE_2 methyl ester) on the inhibition of pentagastrin-induced (0.6 µg/kg-hr, i.v.) gastric secretion were compared in 13 fasted healthy male volunteers. The analogs were administered by stomach tube 45 min prior to the start of pentagastrin infusion. All 3 analogs produced a dose-dependent effect with respect to inhibition of gastric acid secretion ($r = 0.88$-0.98) and duration of response, with an ED_{50} of about 40 µg per subject (60-70% inhibition lasting about 1.5 hr). The inhibition of gastric acid output was primarily due to reduction of volume output which accounted for 80% of the effect. It is concluded that these prostaglandin analogs are potent inhibitors of gastric acid secretion and that they may eventually be useful in the treatment of peptic ulcer.

0865 EFFECT OF ATROPINE ON HYPOGLYCEMIC RELEASE OF GASTRIN IN MAN. *(E.)* Stadil, F.; Malmstrom, J.; Rehfeld, J. F.; and Miyata, M. (Rigshosp., Copenhagen, Denmark. *Acta Physiol. Scand.* 92(3):391-398, 1974.

The effect of atropine on the insulin-produced hypoglycemic release of gastrin was studied in 9 fasted healthy male and female humans. Subjects served as their own controls and gastric acidity, acid output and serum gastrin levels were determined following injection of insulin (0.2 IU/kg i.v.) either alone or preceeded by 25 min with an injection of atropine (30 µg/kg im). Atropine alone had no effect on blood glucose or basal serum gastrin levels. Atropine caused a 60% reduction in peak gastric acid concentration and almost abolished acid output in response to insulin (about 75 mEq H^+/liter and 5 mEq H^+/15 min, resp). Serum gastrin levels following insulin injection reached a maximum (62 pgEq/ml) within 1 hr and remained elevated for 2-3 hr. Atropine caused a further stimulation of serum gastrin which showed a biphasic pattern with a second late increase (81 pgEq/ml) beginning 2 hr after insulin in 4 of the 9 subjects. The differences in gastrin response in the first hr after insulin injection were insignificant. The fact that the gastrin response to hypoglycemia was not decreased by atropine suggests that insulin-induced hypoglycemia caused release of gastrin independently of cholinergic excitation.

0866 CONDITIONING OF GASTRIC SECRETION BY EPINEPHRINE IN RATS. *(E.)* Guha, D.; Dutta, S. N.; Pradhan, S. N. (Howard Univ. Coll. Med., Washington, D.C.). *Proc. Soc. Exp. Biol. Med.* 147(3):817-819, 1974.

The conditioning of gastric secretion in response to a 30 sec, 3000 Hz tone was studied in male and female Wistar rats with gastric cannulas using epinephrine (0.2 mg/kg i.v.) as the unconditioned stimulus. Epinephrine caused a marked decrease in gastric fluid secretion (0.735 ml/hr) compared to saline-injected controls (1.495 ml/hr). In the hour preceeding epinephrine injection, however, a paradoxical increase was seen in the rate of gastric fluid secretion of the experimental group (1.777 ml/hr *vs* 1.509 ml/hr in controls) which was similar to the previously reported increase in salivation seen as a conditioned response to atropine. These results are attributed to an adaptive compensatory mechanism which anticipates the marked inhibition of gastric fluid release induced by epinephrine. The ability to condition this peripheral effect of epinephrine is not in agreement with Astrup's concept which holds that only centrally-mediated effects are conditionable.

0867 GASTRIN-INDUCED HYPOCALCEMIA IN THYROPARATHYROIDECTOMIZED RATS. *(E.)* Schulak, J. A.; Kaplan, E. L. (Univ. Chicago, Pritzker Sch. Med., Ill.). *Metabolism* 23(12):1103-1106, 1974.

The influence of i.v. gastrin injection (6-51 µg/rat) on serum calcium concentration was studied in fasted intact and thyroparathyroidectomized male Holtzman rats. Baseline calcium levels were 10.8-11.1 mg% for intact and 7.3-8.7 mg% for thyroidectomized rats. Thyroid intact rats showed a significant hypocalcemic response 30 min after injection of 12, 25 or 50 µg of synthetic human gastrin or of 12 or 25 µg porcine gastrin. The response was dose-dependent, dropping to 8.7 mg% and 9.2 mg% for human and porcine hormones, resp. Serum calcium returned to normal after 60 min in all animals. Significant transient hypocalcemic responses were also seen in thyroidectomized animals 30 min after injection of 25 µg synthetic human gastrin or 12 or 25 µg porcine gastrin with values of 7.3, 6.5 and 5.7 mg%, resp. These results suggest that gastrin-induced hypocalcemia is not solely dependent upon thyroid calcitonin release and action.

0868 THE INFLUENCE OF RESECTION OF THE SMALL INTESTINE ON GASTRIN LEVELS. *(E.)* Junghanns, K.; Kaess, H.; Dorner, M.; Encke, A. (Univ. Hosp., Heidelberg, Germany). *Surg. Gynecol. Obstet.* 140(1): 27-29, 1975.

The effects of small intestinal resection on serum gastrin levels were studied in 16 dogs with or without denervated gastric pouches. Fasting serum gastrin levels were significantly ($P < 0.05$) higher following resection (101.0 pg/ml) than prior to resection (87.6 pg/ml). Serum gastrin increase in response to feeding was significantly increased only in dogs with innervated Pavlov pouches which had their proximal small bowel resected. Levels reached about 155 pg/ml (1.5-fold greater than control) after 1.5 to 2 hr. Elevated serum gastrin in response to a meal was not so great (about 140 pg/ml, about 1.2-fold greater than control) in animals with resected distal small bowels. Serum gastrin levels were lower in the renal vein than in the renal artery and were lower in the portal vein than in the peripheral blood. These results suggest that the proximal small bowel, as well as the liver and kidney, plays an important role in the inactivation of endogenous gastrin.

0869 CORRELATION BETWEEN SERUM GASTRIN CONCENTRATION AND RAT STOMACH HISTIDINE DECARBOXYLASE ACTIVITY. *(E.)* Hakanson, R.; Kroesen, J. H.; Liedberg, G.; Oscarson, J.; Rehfeld, J. F.; Stadil, F. (Dep. Pharmacol., Univ. Lund, Sweden). *J. Physiol. (Lond.)* 243(2):483-498, 1974.

Serum gastrin concentration and stomach oxyntic mucosa histidine decarboxylase activity were measured simultaneously in fed or fasted adult male Wistar rats which had undergone 1 of the following surgical procedures: 1) vagal denervation with pyloroplasty, 2) antrectomy, 3) antral exclusion, 4) portacaval shunt with or without antrectomy, or 5) sham operation. In addition, some animals received injections of pentagastrin (500 µg/kg s.c.), insulin (2.5 or 10 U/kg s.c.), atropine (10 mg/kg i.p.), or hexamethonium (20 mg/kg i.p.) at various times prior to sacrifice. Freely fed sham-operated rats had much higher serum gastrin levels (153.0 pgEq/ml) and higher oxyntic mucosal histidine decarboxylase activity (35.9 pM CO_2/mg hr) than did fasted, sham-operated animals (35.1 pgEq/ml) and 5.8 pM CO_2/mg hr, resp.). In fasted antrectomized rats the serum gastrin concentrations were low (10.8) although in freely fed antrectomized rats it was the same as in fasted sham-operated rats. Enzyme activity was low (6.1-8.5) in both fasted and fed antrectomized rats. Vagotomized fasted rats had high serum gastrin (119) and high enzyme activity (67.8) whereas vagotomized fed rats had higher gastrin levels (229.2) but comparable enzyme activities. Highest gastrin levels and enzyme activities were seen in fasted antrally excluded rats (343 and 162.2, resp). Analysis by the method of least squares showed a positive correlation between serum gastrin concentration and mucosal histidine decarboxylase activity ($r = 0.69$). Insulin (10 U/kg) produced a slow, progressive increase in serum gastrin concentration in fasted, sham-operated rats which reached a peak (120 pgEq/ml) after 4 hr. An increase in enzyme activity paralleled the gastrin increase but reached a peak (80 pM CO_2/mg hr) 1 hr later. Portacaval-shunted, fasted rats showed a marked activation of gastric histidine decarboxylase following insulin injection which was a least 5 times greater than that in non-shunted rats; serum gastrin was only slightly higher. Shunted rats no longer responded to feeding with elevated enzyme activity following antrectomy indicating that the enzyme-activating agent was of antral origin. In shunted rats, pentagastrin produced an increase in enzyme activity to a level 5 times that of intact rats which was not reduced by antrectomy. These studies failed to provide evidence for the existence of a physiologic

mediator other than gastrin that activates histidine decarboxylase following feeding, vagal stimulation or inhibition of gastric acid secretion.

0870 GASTRIC ORGAN CULTURE: A MODEL FOR REEPITHELIALIZATION. *(E.)* Hayashi, T.; Papla, B.; Stemmermann, G. N. (Kuakini Hosp., Honolulu, Hawaii). *Am. J. Pathol.* 78(1):23-32, 1975.

The cell kinetics of formation of a neoepithelial layer on the cut surfaces of human pyloric antral gastrectomy specimens was studied in organ culture. Light and electron microscopic studies showed that a layer of single epithelial cells began to cover the cut ends of the muscularis mucosae as early as 6 hr after the start of organ culture and that the entire reverse surface of the specimen was epithelialized within 24 hr. Cell outgrowth occurred from normal antral mucosa, from areas of atrophic gastritis and from areas of metaplasia with cells at all levels of differentiation participating in the process. The neoepithelial cells did not form on a basement membrane but rather aligned on collagen fibers or on amorphous interstitial matrix where there were fragments of membranes of disrupted cells and free subcellular organelles. Autoradiographs of sectioned gastric wall pre-labeled with ^{3}H-thymidine showed labeling in about 2% of the neoepithelial cells after 18-20 hr without any definite pattern or distribution of labeled cells; no mitotic figures were observed. A zone of cell extrusion formed in the center of the newly reepithelialized surface; cells in this region showed 3 types of change which appeared to preceed exfoliation: shrinkage necrosis, apoptosis and ballooning of the intercellular space. These findings suggest that differentiation within the neoepithelial cell layer is not the result of maturation during migration and that exfoliation is initiated by cell-cell contact and not by changes in the supporting lamina propria.

0871 GASTRIC AND PANCREATIC RESPONSES TO MEALS VARYING IN pH. *(E.)* Dembrinski, A.; Konturek, S. J.; Thor, P. (Med. Acad., Krakow, Poland). *J. Physiol. (Lond.)* 243(1):115-128, 1974.

Seven chronic gastric and pancreatic fistula cats were used to study the rates of gastric acid and pancreatic secretion in response to a test meal of 10% peptone at various pH values (1.0 to 5.0). Intragastric pH was adjusted by titrating with sodium bicarbonate. Introduction of peptone at pH 5.0 at a relative intragastric pressure of 0 cm resulted in an abrupt increase in gastric acid secretion (1.5 mEq H^+/15 min) which increased still more with increasing pressure up to 25 cm (2.0 mEq H^+/15 min) a value equal to 125-130% the maximum response seen with pentagastrin (16 µg/kg/M) or histamine (320 µg/kg/hr). Pancreatic fluid and bicarbonate outputs were also somewhat stimulated reaching a maximum (about 4 ml/15 min and 0.35 mEq/15 min, resp.) of about 50% of that obtained with exogenous secretin (4 U/kg/hr) at about 15 cm pressure and falling thereafter at higher pressures. Pancreatic protein output was, however, stimulated to a greater degree, reaching 85% of the maximum obtained with cerulein (1.6 µg/kg/hr) at 20-25 cm pressure. When gastric response to a peptone meal was studied over a range of pH values (1.0-5.0), increased acid production occurred at pH values >3.0 with a maximum at pH 4.0 (about 1.7 mEq H^+/15 min) and only a 30% response at pH 1.0 (about 0.4 mEq H^+/15 min). Pancreatic secretory volume and bicarbonate output were, however, stimulated (70% of maximal secretion seen at pH 3.0) at pH values which inhibited gastric acid secretion. The changes in gastric and pancreatic secretion response to a peptone meal at pH 5.0 were reproduced by administration of 1.0 U/kg/hr secretin suggesting that in the cat secretin may be responsible for the gastric inhibitory and pancreatic stimulatory mechanism activated during normal digestion of food.

0872 CONTROL OF GASTRIC ACID SECRETION BY HISTAMINE H_2-RECEPTOR ANTAGONISTS: A PHARMACOLOGICAL DREAM COME TRUE? *(E.)* Ivey, K. J. (R. Prince Alfred Hosp., Camperdown, Australia). *Aust. NZ J. Med.* 4(5):521-524, 1974.

0873 HISTAMINE-STIMULATED HYDROGEN ION SECRETION BY IN VITRO PIGLET GASTRIC MUCOSA. *(E.)* Forte, J. G.; Forte, T. M.; Machen, T. E. (Inst. Anim. Physiol., Cambridge, England). *J. Physiol. (Lond.)* 244(1):15-31, 1975.

0874 CONTROL FACTORS IN THE RELEASE OF GASTRIN BY DIRECT ELECTRICAL STIMULATION OF THE VAGUS. *(E.)* Smith, C. L.; Kewenter, J.; Connell, A. M.; Ardill, J.; Hayes, R.; Buchanan, K. (Univ. Cincinnati Med. Cent., Ohio). *Am. J. Dig. Dis.* 20 (1):13-22, 1975.

0875 INTRAGASTRIC PRESSURE/VOLUME RELATIONSHIP AND GASTRIN RELEASE AFTER DISTENTION OF SEPARATE PARTS OF THE STOMACH. *(E.)* Stadaas, J.; Schrumpf, E. (Ulleval Univ. Hosp., Oslo, Norway). *Scand. J. Gastroenterol.* 9(8):781-785, 1974.

0876 GASTRIC SECRETORY INHIBITION BY CERTAIN METHYL ANALOGS OF PROSTAGLANDIN E_2 FOLLOWING INTESTINAL ADMINISTRATION IN MAN. *(E.)* Nylander, B.; Robert, A.; Andersson, S. (Karolinska Hosp., Stockholm, Sweden). *Scand. J. Gastroenterol.* 9(8):759-562, 1974.

0877 THE PEPSINS OF HUMAN GASTRIC JUICE. SEPARATION BY ELECTROPHORESIS AND CHROMATOGRAPHY; THE INFLUENCE OF GASTRIC DISEASE AND AMBIENT pH. *(E.)* Whitecross, D. P.; Clarke, A. D.; Piper, D. W. (R. North Shore Hosp., Sydney, Australia). *Scand. J. Gastroenterol.* 9(8):711-717, 1974.

0878 SOLUBLE AND BOUND PROTEIN KINASES OF RABBIT GASTRIC SECRETORY CELLS. *(E.)* Ray, T. K.; Forte, J. G. (Dep. Physiol. Anat., Univ. California, Berkeley). *Biochem. Biophys. Res. Commun.* 61(4):1199-1206, 1974.

See also, 0805, 0824, 0836.

SECRETION AND METABOLISM

Pancreas

0879 AN ENZYME WITH COLLAGENOLYTIC ACTIVITY FROM DOG PANCREATIC JUICE. *(E.)* Takahashi, S.; Seifter, S. (Albert Einstein Coll. Med., Yeshiva Univ., Bronx, N.Y.). *Isr. J. Chem.* 12(1/2):557-571, 1974.

The purification and characterization and radioactive assay of a collagenase isolated from the pancreatic juice of dogs prepared with pancreatic fistulae and stimulated to secrete the enzyme by injection of high quality pancreozymin is described. The purification steps included the use of affinity chromatography employing a Sepharose-collagen column. The isolated enzyme had relatively high specific activity, was able to digest extracted, undenatured collagen to peptides of relatively small size, and could act on so-called insoluble beef tendon collagen. The canine pancreatic collagenase was inhibited by diisopropylphosphorofluoridate in relatively high concentrations, cysteine, glycoproteins, human serum, and to a small extent, by soybean trypsin inhibitor. It is suggested that just as carnivorous mammals have evolved with a specific elastase homologous with trypsin and chymotrypsin, they also have evolved in some instances with a specific collagenase; its homology with the other serine proteases is not yet established, although it appears to have properties intermediate between those of a serine protease and those of most tissue collagenases. A radioactive assay for collagenase was developed using a mild treatment with trypsin, which acts only in those sections of the collagen moiety that do not contain the typical collagen helix, and then reacting this material with tritiated sodium borohydride. This treatment causes the introduction of tritium at the sites of aldehyde and aldol crosslinks that were reduced. Subsequent action of an enzyme with collagenolytic activity releases soluble peptides containing tritium; the extent of proteolytic action may then be measured by the radioactivity of the supernatant.

0880 THE PANCREATIC SECRETION OF ENZYMES IN RATS TREATED WITH SOYBEAN DIET. *(E.)* Folsch, U. R.; Wormsley, K. G. (Dep. Med., Univ. Gottingen, Germany). *Scand. J. Gastroenterol.* 9(7):679-683, 1974.

The secretory response to cholecystokinin-pancreozymin (CCK-PZ) and secretin was studied in male Wistar rats which had been treated with a 20-day course of either raw (RSF) or heated (HSF) soybean flour. Experiments were performed on the rats following a 24-hr fast. A glass cannula was placed in the trachea and pancreatic enzymes collected by perfusing the duodenum with 0.15 M NaCl (7.5 ml/15 min). After basal secretion had been collected for 4 successive 15-min periods, secretin and CCK-PZ were infused i.v. at a rate of 1.65 ml/hr. The dose schedules provided submaximal, maximal, and supramaximal stimulation: 1) 7.5 Ivy U/kg-hr CCK-PZ combined with 0.5 clinical U/kg-hr secretin; 2) 60 Ivy U/kg-hr CCK-PZ combined with 0.5 clinical U/kg-hr secretin; and 3) 240 Ivy U/kg-hr CCK-PZ combined with 0.5 clinical U/kg-hr secretin. Only 1 dose combination was used in each animal. Each infusion of hormones lasted for 3 hr and was studied in 3 animals. After an initial weight loss in both the RSF and HSF groups, the HSF-treated rats gained 2.5 times more weight than the RSF-fed rats during the 20-day period. The basal secretion of amylase ranged from 1-9 mM maltose/15 min/100 g during a 4-hr period. Basal secretion of trypsin could not be detected in either group. The peak rate of secretion of trypsin in both groups of rats was attained in all experiments within the first 75 min of the onset of stimulation. The output of trypsin decreased rapidly after reaching a peak rate of secretion. The pattern of secretion of amylase resembled that of trypsin. The overall biphasic pattern of enzyme secretion of the RSF rats was not different from the control (HSF) rats, and the maximal rate of secretion of trypsin was identical in the 2 groups of rats. Outputs of amylase and trypsin, however, were significantly increased in the RSF animals compared with control in response to the submaximal and supramaximal doses of CCK-PZ, because the secretion of enzymes was better sustained in the RSF rats than in the control rats. It is concluded that despite the very marked histological changes in the pancreas of rats treated with RSF, the pattern of secretory response to stimulants of the hypertrophied gland is not qualitatively different from normal.

0881 NUTRITIONAL AND HORMONAL EFFECTS ON RNA POLYMERASE ENZYME ACTIVITIES IN PANCREAS. *(E.)* Black, Jr., O.; Webster, III, P. D. (VA Hosp., Augusta, Ga.). *Am. J. Physiol.* 227(6):1276-1280, 1974.

The responses of DNA-dependent RNA polymerase I and polymerase II enzyme activities were studied in pigeon pancreas in response to fasting, cholinergic, and hormonal stimulation. Following 72 hr of fasting, polymerase I and II activities, which were measured as incorporation of [^{14}C] uridine triphosphate, decreased by 58% and 89%, resp. Nuclear RNA content decreased 23%. None of these changes was associated with increased RNase activity. In response to bethanechol chloride (2 mg/kg) and cholecystokinin-

pancreozymin (CCK-PZ) (15 U/kg), polymerase I activity increased in a differential manner. Both increased the quantities of 28-S and 18-S RNA species. Enzyme activities in bethanechol chloride treated pigeons increased within 10 min but there was no significant change in polysomal RNA after 50 min. Pancreases from (CCK-PZ) treated pigeons showed increased polysomal RNA within 15 min after maximum enzyme activity was observed. Polymerase II enzyme activities increased with both drugs to a maximum 45 min after administration. The evidence indicates that: 1) the pancreas changes in nuclear ribosomal RNA synthesis as a result of fasting, cholinergic, and hormonal stimulation; 2) the stimulated RNA synthesis resulting from (CCK-PZ) and bethanecol chloride stimulation occurred at a time when protein synthesis was inhibited and energy supply was low; and 3) the pancreas responds to hormonal stimulation more rapidly than other tissues.

0882 SUBCELLULAR CHANGES IN PROTEIN METABOLISM AND MAGNESIUM CONTENT OF THE PANCREAS IN RATS FED TWO LEVELS OF DIETARY PROTEIN AND MAGNESIUM. *(E.)* Greger, J. L.; Schwartz, R. (Coll. Hum. Ecol., Cornell Univ., Ithaca, N.Y.). *J. Nutr.* 104(12):1618-1629, 1974.

The subcellular distribution of Mg^{++} as determined by atomic absorption spectrophotometry and the incorporation of i.v. injected ^{14}C-valine into pancreatic proteins were studied in 120 male Sprague-Dawley rats. Animals were fed a diet for 10 days containing 15 or 30% casein and 50 or 100 ppm Mg^{++} with or without fasting for 24 hr prior to sacrifice. The rates of protein synthesis and secretion were estimated by determining the label present in the acid soluble and insoluble portions of pancreatic microsomal, zymogen and supernatant fractions 5, 45 and 120 min after injection of ^{14}C-valine. Protein specific activity was decreased in the acid soluble microsomal fraction, the acid soluble supernatant fraction and the total mitochondrial fraction of fasted Mg^{++} deficient rats 2 hr after ^{14}C-valine injection. Mg^{++} levels in the zymogen, supernatant, mitochondrial and nuclear plus cellular debris fractions were unaffected by diet content although refeeding resulted in decreased zymogen and supernatant Mg^{++}. Total microsomal Mg^{++} content, however, was significantly greater in both fasted and refed rats fed the 30% casein, 100 ppm Mg^{++} diet than in rats fed 50 ppm Mg^{++} diets or the 15% casein, 100 ppm Mg^{++} diet. Acid soluble microsomal Mg^{++} content was 5- to 10-fold greater than acid insoluble content. These results provide little, if any, evidence of significant shifts in the intracellular distribution of Mg^{++} with changes in diet.

0883 REGULATION OF PANCREATIC ACINAR FUNCTION: EFFECTS OF CYCLIC AMP, DIBUTYRYL CYCLIC AMP, AND THEOPHYLLINE IN VITRO. *(E.)* Haig, T. H. B. (Dep. Surg., Univ. Saskatchewan, Saskatoon, Canada). *Can. J. Physiol. Pharmacol.* 52(4):780-785, 1974.

The role of cyclic AMP in the regulation of pancreatic acinar function was assessed by measuring the effects of exogenous cyclic AMP, dibutyryl cyclic AMP, and theophylline on protein synthesis and amylase secretion in slices of rat pancreas. Pancreas slices from 1 healthy male albino rat were used for each experiment. The slices were placed in a vial with 2 ml of tissue culture medium containing 0.20 μCi/ml of L-leucine-^{14}C plus the stimulant(s) to be tested. Experiments testing cyclic AMP and dibutyryl cyclic AMP were run using 10 min and 160 min incubation times (the former for measuring secretion and the latter measuring synthesis), and 3 concentrations of cyclic nucleotide (2×10^{-3} M, 1×10^{-3} M, and 0.5×10^{-3} M) plus 2 concentrations of pancreozymin (5 U/ml and 2.5 U/ml), as well as a control. Theophylline was assessed using the same 2 incubation times and testing 2 concentrations (10^{-3} M and 10^{-4} M) with and without pancreozymin (5 U/ml and 2.5 U/ml). Six animals were tested in each of the 3 sets of experiments. After the selected incubation time, the medium was pipetted from each vial and assayed for amylase activity, and for L-leucine-^{14}C incorporation into protein. In this test system, the rate at which slices of rat pancreas incorporated leucine into protein did not change as a consequence of treatment with either cyclic AMP or dibutyryl cyclic AMP, nor did the slices alter their rate of amylase secretion. In contrast, rat pancreas slices secreted amylase vigorously in response to pancreozymin, while simultaneously decreasing their mean rate of protein synthesis. Theophylline did not enhance the ability of submaximal doses of pancreozymin to stimulate amylase secretion or to suppress protein synthesis. These results fail to demonstrate that cyclic AMP regulates either the synthesis or secretion of pancreatic digestive enzymes but they do not rule out the possibility.

0884 PANCREATIC ACINAR CELLS: EFFECTS OF LANTHANUM IONS ON AMYLASE RELEASE AND CALCIUM ION FLUXES. *(E.)* Chandler, D. E.; Williams, J. A. (Dep. Physiol., Univ. California, San Francisco). *J Physiol. (Lond.)* 243(3):831-846, 1974.

The effect of La^{+++} on amylase release and Ca^{++} fluxes in mouse pancreatic fragments *in vitro* was studied. Amylase release was increased by 0.1 mM La^{+++} and progressively inhibited by 1.0-10 mM La^{+++}. Non-stimulated and bethanecol stimulated secretion were altered in an identical manner. Inhibition of amylase release was rapid and reversible. Uptake of $^{45}Ca^{++}$ was multiphasic with equilibrium with stable Ca^{++} still not complete after 2 hr. La^{+++} (10 mM) limited uptake of $^{45}Ca^{++}$ to the extracellular space and slightly decreased total Ca^{++} content. Lower concentrations of La^{+++} affected $^{45}Ca^{++}$ uptake and total Ca^{++} content in a biphasic manner which paralleled effects on amylase release. La^{+++} restricted washout of $^{45}Ca^{++}$ to isotope in the extracellular space and abolished the bethanecol-stimulated increase in $^{45}Ca^{++}$ efflux. Uptake of $^{45}Ca^{++}$ into intracellular space was not affected by bethanecol. Tissue ultrastructure and Na^{+} and K^{+} content were not affected by La^{+++}. It is concluded that an influx of extracellular Ca^{++} is not important for triggering of secretion and that La^{+++} may inhibit amylase release by acting on the release process rather than on Ca^{++} influx.

0885 PANCREATIC EXOCRINE SECRETION DURING AND AFTER PREGNANCY. *(E.)* Rosenberg, V.; Rudick, J.; Robbiou, M.; Dreiling, D. A. (Mt. Sinai Sch. Med., City Univ. New York, N.Y.). *Ann. Surg.* 181(1):47-50, 1975.

Pancreatic secretion was evaluated in 8 pregnant mongrel dogs prepared with Thomas duodenal and gastric fistulas, during pregnancy at a time corresponding to the third trimester in humans, during the puerperium, and several months after whelping. Basal secretion increased in volume from 0.85 to 1.87 ml/10 min during pregnancy and to 1.74 ml/10 min during puerperium. Mean bicarbonate concentration of 49.4 mEq/liter rose to 79.5 and 74.6 mEq/liter during pregnancy and in the puerperium, resp. Bicarbonate output was increased by 319% during pregnancy and 211% in the puerperium. The response to exogenous secretin, submaximal and maximal, was unchanged during pregnancy but decreased in the puerperium. Enzyme output in the resting state was increased by 73% during pregnancy and 35% in the puerperium. The responses to cholecystokinin-pancreozymin was markedly increased during pregnancy but not in the puerperium. Enzyme responses to cholecystokinin-pancreozymin during pregnancy were increased not only in output but also in concentration. Although the precise mechanism is not clear, the alterations in pancreatic exocrine function observed here could contribute to the pancreatitis of pregnancy and the puerperium.

0886 GLUCOSE-INDUCED INSULIN SECRETION AND PATTERN OF EXOCRINE PANCREATIC ENZYMES IN THE RAT FOLLOWING ORAL AND PARENTERAL TRYPSIN INHIBITOR ADMINISTRATION. *(E.)* Ihse, I.; Arnesjo, B.; Lundquist, I. (Dep. Surg., Univ. Lund, Sweden). *Scand. J. Gastroenterol.* 9(8):719-724, 1974.

The pancreatic enzymic pattern and endocrine function were compared after long-term oral or i.p. administration of bovine lung trypsin inhibitor to male Wistar rats. One daily dose of trypsin inhibitor was administered orally (100,000 Kallikrein-inhibiting U in 1 ml water by gastric intubation) or i.p. (1 ml 100,000 Kallikrein-inhibiting U/ml) every morning for 3 weeks. After oral administration the wet weight and the protein content of pancreas were increased. The pancreatic trypsinogen increased in a more pronounced way than the amylase and lipase activities. The trypsin-inhibitor-treated group showed a decreased insulin secretory response 3 and 7 min after i.v. glucose administration (0.75 g/kg) and 30 and 60 min after p.o. ingestion of a 30% glucose solution (1 ml/100 g). The glucose tolerance was normal or even increased. No changes in pancreatic wet weight, protein content, enzyme pattern, and endocrine pancreatic function were found after i.p. trypsin inhibitor administration. The results support the hypothesis that the effects on exocrine and endocrine rat pancreatic functions of trypsin inhibitor treatment require the mediation of the gastrointestinal tract.

0887 THE INFLUENCE OF REDUCED PANCREATIC BLOOD FLOW ON STIMULATED EXOCRINE PANCREATIC FUNCTION. *(E.)* Laszcz, M.; Zimmermann, G.; Umlauft, M.; Boeckl, O. (Ludwig Boltzmann Inst. Exp. Surg., Salzburg, Austria). *Eur. Surg. Res.* 6(1):59-60, 1974.

See also, 0871, 0903, 1106.

SECRETION AND METABOLISM

Hepatobiliary Tract

0888 STUDIES ON THE PATHOGENESIS OF EXPERIMENTAL CHRONIC ACTIVE HEPATITIS IN RABBITS: II. DEMONSTRATION OF IMMUNOGLOBULIN ON ISOLATED HEPATOCYTES. *(E.)* Hopf, U.; Meyer Zum Buschenfelde, K. H. (2nd Dep. Intern. Med., Univ. Mainz, Germany). *Br. J. Exp. Pathol.* 55(5):509-513, 1974.

Immunofluorescence studies were conducted on hepatocytes isolated from white New Zealand rabbits with cirrhosis and chronic active hepatitis experimentally induced by repeated immunization (25-41 injections over 79-143 week) with human liver protein (5-15 mg i.p. each injection) plus complete Freund's adjuvant. Immunoglobulin could be demonstrated on the hepatocyte membranes of 8 of 9 animals with chronic active hepatitis. The 1 animal with cirrhosis showed no membrane fixed immunoglobulin. Of 8 animals without liver lesions which had been immunized with both human and rabbit liver protein, only 1 showed a weak membrane fluorescence. The presence or absence of liver lesions had no effect on serum antibody titers against liver specific proteins as tested for by passive hemagglutination and gel diffusion. A positive skin test to rabbit liver protein correlated with the development of liver lesions. These results are consistent with an antibody mediated lymphocyte toxicity as mediator for the development of chronic active hepatitis under these conditions.

0889 MORPHOLOGICAL STUDIES ON THE RAT LIVER AND A BIOCHEMICAL ANALYSIS OF THE SERUM FOLLOWING EXPERIMENTAL LIGATURE OF THE THORACIC DUCT. *(E.)* Cremer, H.; Muller, N.; Bechelsheimer, H. (Inst. Pathol., Univ. Bonn, Germany). *Lymphology* 7(2):69-80, 1974.

Light microscopic studies and serum determinations of various liver enzyme activities were conducted on

livers of 308 white male and female Sprague-Dawley rats following induction of hepatic lymphedema by ligature of the thoracic duct. Following thoracic duct ligations, the spaces of Disse and Mall as well as the lymphatic vessels in the portal tracts and the lymphatics along the hepatic veins became dilated. Necrosis of the hepatocytes along the periphery of the liver lobules were seen and there was evidence of formation of lymphobiliary fistulas. These morphological changes regressed somewhat after 12-15 days. Within the 1st day following thoracic duct ligation, serum enzyme activities as determined photometrically showed a 4-fold increase in LDH and approximately 3- and 2-fold increases in SGOT and SGPT, resp. Serum alkaline phosphatase activity showed a 60% decrease during the 1st day and remained depressed throughout the 90 days of the experiment. Serum triglyceride levels also decreased by 60% the 1st day, returned to normal, and showed another 50% decrease on day 15. Total lipid and cholesterol values did not differ significantly from pre-experimental values. It was concluded that the observed morphological and biochemical changes were secondary to hepatic lymphedema and that the implications of these findings may be applied in the management of various liver diseases (e.g., liver transplants and carcinomatous lymphangiosis) in which hepatic lymphatic blockage may be significant.

0890 UNUSUAL LYSOSOMES IN HAMSTER HEPATOCYTES. *(E.)* Nehemiah, J. L.; Novikoff, A. B. (Albert Einstein Coll. Med., Yeshiva Univ., Bronx, N.Y.). *Exp. Mol. Pathol.* 21(3):398-423, 1974.

Light and electron microscopic studies were conducted on the hepatic lysosomes from male and female Syrian golden hamsters fed either a normal or a cholesterol-supplemented diet (2%) for up to 10 months. Unlike the usual electron dense lysosomes usually observed in mammalian hepatocytes, some of these displayed a relatively large lipid inclusion which could be stained with oil red O and were also acid phosphatase positive. The acid phosphatase activity was limited to the area immediately around the lipid inclusion and, in most cases, to a region at one pole of the lysosome which was typically electron-dense. These lipid-containing lysosomes were increased in the livers of male and female animals maintained on the cholesterol-supplemental diet, appearing first in periportal hepatocytes but later also in centrilobular hepatocytes. Frozen liver sections from these hamsters showed the characteristic "Maltese cross" birefrigence of cholesterol-containing lipids. Liver cholesterol levels as determined by chemical means rose rapidly following a single feeding of the cholesterol-supplemented diet and reached levels 30-fold greater than controls after 3 weeks. Electron microscopic studies suggested that the lipid inclusions were forming in the Golgi-associated endoplasmic reticulum or in the neighboring endoplasmic reticulum proper since their membranes appeared to be continuous with those of the lipid-containing lysosomes. Removal from the cholesterol-supplemented diet resulted in the gradual disappearance of the electron-lucent lipid with the lysosomes coming to resemble lipofuscin granules. Thus, hamster liver may serve as a model for inherited disorders of lipid metabolism and other diseases in which electron-lucent lipid accumulates within lysosomes.

0891 LIVER REGENERATION AFTER PORTACAVAL TRANSPORTATION IN RATS. *(E.)* Lee, S.; Broelsch, C. E.; Flamant, Y. M.; Chandler, J. G.; Charters, III, A. C.; Orloff, M. J. (Sch. Med., Univ. California San Diego, La Jolla). *Surgery* 77(1):144-149, 1975.

Liver regeneration was studied in 40 partially hepatectomized inbred male Lewis rats which had previously undergone either sham operation, an end-to-side portacaval shunt or a portacaval transposition. Six criteria were used to evaluate regeneration: wet weight, dry weight, protein content, mitotic index, percent labeled nuclei following ^{3}H-thymidine injection, and rate of incorporation of ^{3}H-thymidine into liver DNA. By all criteria except mitotic index, active regeneration occurred in all experimental groups, although somewhat less rapidly than that of the sham operated controls. Mitotic indices were significantly greater in livers of animals with portacaval shunts than in those with the added benefit of a substitute systemic venous flow. These results thus indicate that there is no added regenerative benefit afforded by substitution of a systemic venous flow for portal flow to the liver.

0892 ADENINE NUCLEOTIDE METABOLISM IN REGENERATIVE, ATROPHIC, AND NECROTIZING PROCESSES OF THE LIVER. *(E.)* Ozawa, K.; Takeda, H.; Tamaoka, Y.; Nambu, H.; Kamiyama, Y.; Honjo, I. (Kyoto Univ. Fac. Med., Japan). *Gastroenterology* 67(6):1225-1230, 1974.

Mitochondrial adenine nucleotide metabolism was studied during regenerative, atrophic, and necrotizing processes of liver in mammals subjected to ligation of the left portal vein or the common bile duct. Phosphorylase activity per unit of cytochrome ($a+a_3$) of isolated liver mitochondria from ligated lobes was decreased to about 50% of the sham-operated control value within 12 hr of ligation and remained relatively constant for the remaining 5.5 days of the experiment. The energy charge ($ATP+\frac{1}{2}ADP/ATP+ADP+AMP$) decreased by about 40% immediately following ligation but returned to normal on the 4th day. Measurement of hepatocellular adenine nucleotide concentrations by an enzymatic method showed that ATP remained unchanged but ADP, AMP and total adenine nucleotide concentrations decreased significantly following common duct ligation; ADP, AMP and total adenine nucleotide concentrations were increased in the non-ligated lobes. Phosphorylase specific activity was the same in normal liver mitochondria from man, monkey, dog and rabbit but was decreased up to 50% in monkey, dog and rabbit folowing portal vein ligation; phosphorylase specific activity from the non-ligated lobes was about 45% higher than that from normal liver. Phosphorylase specific activity was decreased to about 35% and adenine nucleotide concentration fell to 50% or less than that of normal liver in severely injured livers of icteric rabbits subjected to common bile duct ligation. Most of these animals died of hepatic insufficiency within 3 days. These results indicate that phosphorylase specific activity increases in regener-

ating liver and decreases in atrophic (especially necrotizing) processes. The major feature of the regenerative process appears to be an increase in the capacity for aerobic metabolism.

0893 ROLE OF INSULIN AS A PORTAL FACTOR IN MAINTAINING THE VIABILITY OF LIVER. *(E.)* Ozawa, K.; Yamada, T.; Honjo, I. (Kyoto Univ. Fac. Med., Japan). *Ann. Surg.* 180(5):716-719, 1974.

The effect of insulin on hepatic mitochondrial oxidative phosphorylation was studied in adult male Wistar rats subjected to ligation of the left portal vein. Simultaneous s.c. administration of insulin (2.5 U/kg) and intragastric intubation of glucose (400 mg/200 gm) had no effect after 2 hr on the respiratory control ratio, state 3 respiration, P:O ratio and phosphorylase activity of normal hepatic mitochondrial preparations from non-ligated rats. Following ligation of the left portal vein, liver mitochondrial phosphorylase activity decreased within 12 hr from 110 to 60 mM ATP/mg protein/min with glutamate as substrate. Administration of insulin (0.25-2.5 U/kg) to ligated rats resulted in up to more than a 2-fold increase in the rate of hepatic mitochondrial phosphorylation. Kinetic studies of phosphorylase activity in ligated rat livers following s.c. injection of 2.5 U/kg insulin showed a rapid stimulation within 30 min to a value 200% of control within 2 hr ($P < 0.005$) with a subsequent fall to subnormal levels. The respiratory control ratio, state 3 respiration and P:O ratio were maximally increased in parallel with the increased phosphorylase activity. Respiratory enzyme concentrations, however, remained unchanged. It is suggested that insulin may mediate mitochondrial metabolism by affecting levels of a previously reported portal vein factor which can stimulate mitochondrial oxidative phosphorylation.

0894 ALBUMIN, FIBRINOGEN AND TRANSFERRIN SYNTHESIS IN ISOLATED RAT HEPATOCYTE SUSPENSIONS: A MODEL FOR THE STUDY OF PLASMA PROTEIN SYNTHESIS. *(E.)* Jeejeebhoy, N.; Ho, J.; Greenberg, G. R.; Phillips, M. J.; Bruce-Robertson, A.; Sodtke, U. (Dep. Med., Univ. Toronto, Canada). *Biochem. J.* 146(1): 141-155, 1975.

A model system for studying synthesis of albumin, fibrinogen and transferrin has been established using suspension cultures of hepatocytes from healthy Wistar rats. Optimum culture conditions for survival and protein synthesis included an enriched medium using 17.5% heat-inactivated horse serum, constant stirring and an O_2 tension between 18.7×10^3 and 26.7×10^3 Pa. Solid-phase radioimmunoassay showed that the rates of synthesis of albumin, fibrinogen and transferrin were 0.32, 0.12 and 0.097 mg/hr/g of hepatocytes, resp. These rates were maintained for the first 12 hr after which they decreased to lower levels which were maintained for up to 48 hr. Albumin synthesis was decreased but fibrinogen synthesis was unchanged in relatively amino acid- and glucose-deficient medium. ATP concentration and the RNA:DNA ratio of cells incubated up to 12 and 24 hr, resp., were comparable to those of hepatocytes *in vivo*. Estimates of protein synthesis of hepatocytes maintained for 48 hr in medium supplemented with a hormone mixture consisting of glucagon (0.75 ng/ml), cortisol (0.25 µg/ml), triiodothyronine (0.09 µg/ml) and bovine growth hormone (0.5 µg/nl) increased fibrinogen synthesis by over 2-fold but had no effect on albumin or transferrin synthesis. Addition of insulin (20 µU/ml) increased albumin and transferrin synthesis by about 35% but did not further stimulate fibrinogen synthesis. This system thus responds in a manner comparable to that *in vivo* and in long-term perfused whole liver and may thus prove useful for *in vitro* testing of hepatotropic factors.

0895 EFFECTS OF OCTANOATE AND OLEATE ON ENERGY METABOLISM IN THE PERFUSED RAT LIVER. *(E.)* Debeer, L. J.; Mannaerts, G.; De Schepper, P. J. (Sch. Med., Cathol. Univ. Louvain, Belgium). *Eur. J. Biochem.* 47(3):591-600, 1974.

Livers from fasted male Wistar rats (100-120 g) were perfused with octanoate or oleate added as a single dose after an equilibration period of 28 min and the perfusions stopped at 35 or 60 min. Octanoate as well as oleate induced a net decrease in ATP and an increase in AMP content of the liver. Total adenine nucleotides were unaltered. ATP:ADP ratios were lowered while the adenylate kinase mass-action ratio increased. In other experiments, livers from fasted rats were perfused without substrate for 58 min, after which substrate was added to obtain an initial concentration of 1 mM or 5 mM octanoate, 1 mM oleate or 10 mM lactate. The Krebs cycle activity was suppressed by both fatty acids and enhanced by lactate. Oxidation at C-3 was strongly stimulated by the fatty acids and was unaffected by lactate. An apparent ADP:O ratio of 2.7 was obtained after lactate addition, indicating a tightly coupled oxidative phosphorylation for the liver preparations used. Octanoate and oleate gave extremely low ratios of near one and near zero resp. Perfusion with oligomycin (1 mg/100 ml perfusion medium) caused a severe drop in oxygen uptake by the liver, which was unaltered after addition of fatty acids. Oligomycin added after the fatty acids caused an immediate fall in oxygen uptake to the level observed with oligomycin alone. 2,4-Dinitrophenol (1 mM) was able to stimulate the oligomycin-depressed respiration, both in the absence and in the presence of fatty acids. Under these experimental conditions, therefore, the fatty acids had no uncoupling effect and microsomal fatty acid oxidation was minimal. Incorporation of $[^3H]$leucine (10 µCi added to the perfusion medium) into proteins in liver and medium remained unchanged after addition of fatty acids, indicating that the low apparent ADP:O ratios are not the result of an enhanced synthesis of proteins.

0896 AGE-DEPENDENT INDUCTION AND REPRESSION OF RAT LIVER MICROSOMAL HYDROXYLASE SYSTEMS BY ESTRADIOL. *(E.)* Gustafsson, J. A.; Ingelman-Sundberg, M. (Karolinska Inst., Stockholm, Sweden). *Biochim. Biophys. Acta* 354(2):172-181, 1974.

The activities of the hepatic microsomal 2α-, 2β-, 18- and 7α-hydroxylase systems active on 5α[4-^{14}C]-androstane-3α,17β-diol were studied in male and female Sprague-Dawley rats. Animals were castrated and spayed at 14, 24, 35 and 45 days of age, treated for 5 days with 500 μg of estradiol benzoate i.m./kg and killed 6 days after gonadectomy. The hepatic microsomal 15β-hydroxylase enzyme system active on 5α-[1,2-^{3}H]androstane-3α,17β-diol 3,17-disulphate was also measured in these animals as well as in an additional group of animals that were gonadectomized at 56 days of age, treated with estradiol benzoate and killed at 62 days of age. The hydroxylase systems active on the free steroid substrate were all relatively unresponsive towards estradiol in the developing rat, in contrast to the strong effects on hepatic hydroxylase activities previously noted following treatment of adult male rats with estradiol. On the other hand, the 15β-hydroxylase system active on disulphurylated 5α-androstane-3α,17β-diol was inducible in both male and female rats of 41 and 51 days of age and in male rats of 61 days of age. These results may be explained at least in part by variations in concentration, during different stages of development, of the cytoplasmic receptor and/or nuclear acceptor proteins specific for estradiol.

0897 LIVER ENZYMES IN THE ECK FISTULA RAT. I. UREA CYCLE ENZYMES AND TRANSAMINASES. *(E.)* Colombo, J. P.; Herz, R.; Bircher, J. (Dep. Clin. Pharmacol., Univ. Bern, Switzerland). *Enzyme* 14:353-365, 1972/73.

Male Sprague-Dawley rats were sacrificed at the end of 10, 20, and 30 days after portacaval shunt. Two non-operated control animals were always sacrificed at the same time. The liver was removed immediately, frozen, and then homogenized for the assessment of enzyme activities; the protein content of the liver homogenate was also determined. Blood for ammonia determination was drawn from the orbital venous plexus and ammonia content measured immediately using an ion-exchange resin and the Berthelot color reaction. Three typical responses in the activities of the urea-cycle enzymes were observed: type I, normal or slightly decreased enzyme activity/g liver, but decreased/total liver mass (ornithine carbamyltransferase, argininosuccinate synthetase [rate-limiting step of the urea cycle], arginase, glutamate pyruvate, and oxaloacetate transaminase, glutamate dehydrogenase); type II, increased enzyme activity/g liver, but decreased/total liver mass (carbamylphosphate synthetase); and type III, increased enzyme activity/g liver, but normal/total liver mass (argininosuccinate cleavage enzyme). The findings suggest that shunt-induced liver "atrophy" and consequent reduction in functional-enzyme content represent at least one of the factors contributing to the rise in peripheral blood-ammonia concentration and potentially to the development of features interpreted as "portal-systemic encephalopathy".

0898 EFFECT OF MICROSOMAL ENZYME INDUCERS ON THE LIVER CHANGES PRODUCED BY COMMON BILE-DUCT LIGATURE. *(E.)* Solymoss, B.; Zsigmond, G. (Dep. Pathol., Univ. Montreal, Quebec, Canada). *Proc. Soc. Exp. Biol. Med.* 147(2):430-433, 1974.

The biochemical and morphological changes in the livers of female ARS/Sprague-Dawley rats were studied following ligation of the common bile duct. Groups of 8 animals each served as untreated controls, as ligated controls, or else were ligated and then received (3 days/week) by gastric intubation, spironolactone (84 mg/kg), pregnenolone-16α-carbonitrile (PCN, 68.7 mg/kg) or phenobarbital (50.8 mg/kg) between the 5th and 39th day of the experiments. At the end of the 39th day, the livers of all ligated animals weighed 2-3 times more than those of untreated controls. Electron microscopic analysis also showed bile duct proliferation and hyperplasia of the smooth endoplasmic reticulum. Microsomal protein content of livers from ligated controls was significantly less (20.84 mg/kg) than that from untreated controls (30.36 mg/g). Microsomal protein content of livers from PCN- or phenobarbital-treated ligated rats was, however, greater (23.29 and 24.40 mg/kg, resp.) than that of the ligated controls. Bile duct ligation resulted in an 8-fold decrease in microsomal cytochrome P-450 content, a 2-fold decrease in NADPH cytochrome C reductase specific activity, and a 3.5-fold decrease in ethylmorphine N-demethylase specific activity. Treatment with PCN, however, resulted in an increased cytochrome P-450 content, restored NADPH cytochrome C reductase activity to normal, and increased ethylmorphine N-demethylation. Spironolactone and phenobarbital produced less pronounced effects. PCN also stimulated hepatic bilirubin-UDP-glucuronyl transferase activity compared to untreated controls. Such activity was decreased in ligated but otherwise untreated rats. These results confirm previous findings in which long-term ligation of the common bile duct produced functional and structural alterations of the hepatic endoplasmic reticulum. Of the microsomal enzyme inducers tested, PCN was the most effective in reversing these changes.

0899 STIMULATION OF THE DEVELOPMENT OF THE HEPATIC EXCRETORY MECHANISM FOR OUABAIN IN NEWBORN RATS WITH MICROSOMAL ENZYME INDUCERS. *(E.)* Klaassen, C. P. (Univ. Kansas Med. Cent., Kansas City). *J. Pharmacol. Exp. Ther.* 191(2):212-218, 1974.

To determine whether various microsomal inducers would stimulate an early development of the hepatic mechanisms responsible for removal of ouabain from plasma, and its release into the bile, newborn rats (Sprague-Dawley, 1-31 days), were pretreated with these substances for 4 days. The inducers used were phenobarbital sodium (33 or 50 mg/kg), spironolactone (75 mg/kg), pregnenolone-16α-carbonitrile (PCN, 75 mg/kg), or 3-methylcholanthrene (20 mg/kg), each of which was given i.p. in propylene glycol solution. Control rats were given only propylene glycol (5 ml/kg). On the fifth day, 4 mg/kg ^{3}H-labeled ouabain octahydrate was administered i.v. After 15 min a blood sample was taken *via* cardiac puncture. The rats were then sacrificed, and the liver and small intestine removed. The small intestine was homogenized

and assayed for ouabain by means of an Eberbach microadapter. Liver samples were counted by liquid scintillation spectrometer. To determine the effect of the inducers on toxicity, rats were injected with them on days 6, 7, 8, and 9, and given ouabain on day 10. It was found that phenobarbital sodium, PNC, and spironolactone all decreased the plasma ouabain concentration in all age rats. They also decreased the ouabain concentration in the livers of rats 25 days and older, but increased them in younger rats (approximately 15 days old). The 3-methylcholanthrene did not decrease plasma concentration, and tended to increase liver concentration. It did not alter ouabain toxicity, while the other 3 inducers decreased it significantly in 10 day old rats. It is concluded that in the newborn rat, some microsomal enzyme inducers will increase biliary excretion of ouabain by enhancement of its transfer from plasma to liver. In rats over 25 days old, the major effect is enhancement of the transfer from hepatocyte to bile.

0900 EFFECT OF URIDINE-DIPHOSPHATE-GLUCOSE ON LIVER GLUCURONYL TRANSFERASE ACTIVITY IN NORMAL AND IN FASTING RATS. *(E.)* Okolicsanyi, L.; Scremin, S. (Univ. Polyclin., Padova, Italy). *Enzyme* 14:366-371, 1972/73.

Six Sprague-Dawley rats received 20 mg/kg uridinediphosphate glucose (UDPG) in physiological saline i.p. 1 hr before taking liver specimens; in a second group of 6 rats, 20 mg/kg UDPG was administered for 5 days i.p.; and in the third group of 6, 50 mg/kg UDPG were given for 5 days i.p. Another 6 animals served as controls for glucuronyl transferase (GT) activity. At the end of the experimental period, liver specimens were taken under pentobarbitone anesthesia; the GT activity was determined within 48 hr using 4-nitrophenol as substrate. After UDPG treatment GT activity increased significantly, but with 50 mg/kg UDPG the rise of enzyme activity was less important than with 20 mg/kg. The 1-hr treatment did not affect GT activity. In another series of experiments, 6 rats were submitted to a 72 hr fast; 6 other animals were also fasted for 72 hr but were treated for 5 days with 20 mg/kg UDPG (2 days before beginning the fast and during the 72-hr fasting period). Fasting caused an important decrease in GT activity, while enzyme activities did not change significantly in fasting rats treated with UDPG. The mechanism through which UDPG exerts these effects remains to be established.

0901 EVIDENCE FOR AN ACTIVATING-INACTIVATING SYSTEM OF 3-HYDROXY-3-METHYLGLUTARYL-CoA REDUCTASE IN MOUSE LIVER. *(E.)* Berndt, J.; Gaumert, R. (Inst. Biochem., Soc. Radiat. Environ. Res., Munich, Germany). *Hoppe Seylers Z. Physiol. Chem.* 355(7):905-910, 1974.

The activity of 3-hydroxy-3-methylglutaryl-CoA reductase, which catalyzes the first step in cholesterol synthesis, was studied in liver microsomal preparations from male White mice. Crude liver homogenates were found to contain an activating-deactivating system for the enzyme not present in purified microsomal preparations. Preincubation of microsomal preparations with various subcellular fractions followed by *in vitro* assay of microsomal reductase activity showed that the reductase activating system was present only in the cytoplasmic fraction. The ability of trypsin to destroy this activating system established that it is proteinaceous mature. Assay of microsomal reductase activity in livers from mice following 24-hr starvation or feeding of a high-cholesterol diet, two factors known to decrease reductase activity, showed decreased enzyme activation in both cases. The physiologic significance of these findings remains to be determined.

0902 STUDIES OF SERUM LECITHIN-CHOLESTEROL ACYL TRANSFERASE ACTIVITY IN RAT: EFFECT OF VITAMIN E DEFICIENCY, OXIDIZED DIETARY FAT, OR INTRAVENOUS ADMINISTRATION OF OZONIDES OR HYDROPEROXIDES. *(E.)* Takatori, T.; Privett, O. S. (Hormel Inst., Univ. Minn., Austin). *Lipids* 9(12):1018-1023, 1974.

Serum lecithin-cholesterol acyl transferase activity was studied in male Sprague-Dawley rats fed a diet for 9 months depleted of vitamin E or for 10 months containing oxidized fat. In other experiments, the effects of i.v. injected methyl linoleate hydroperoxide and methyl linoleate ozonide (20 mg/100 g) were studied on serum acyl transferase activity. Acyl transferase activity was decreased by vitamin E deficiency from a control value of 778 nM ^{14}C-cholesterol esterified/min/liter to 531 nM/min/liter ($P < 0.02$). Dialuric acid-induced erythrocyte hemolysis was about 30-fold greater in vitamin E deficient than in control rats. Vitamin E deficiency had no effect on serum fatty acid composition, cholesterol, free fatty acid or triglycerides but did significantly elevate serum cholesterol esters from 80.8 (in controls) to 93.6 mg% ($P < 0.05$). Serum acyl transferase activity was significantly increased in rats fed a diet containing 2% oxidized safflower oil (1150 *versus* 785 nM/min/liter) or oxidized menhaden oil (1500 *versus* 651 nM/min/liter). Injection of methyl linoleate hydroperoxide or ozonide caused an immediate 2.5- to 3-fold decrease in acyl transferase activity which returned to normal after 5 hr. Injection of 2-mercaptoethanol, a substance which counteracts sulfhydryl-blocking agents, 5 min after injection of the hydroperoxide or ozonide partially reversed the inhibition. Thus, the inhibitory effect of hydroperoxides and ozonides on lecithin-cholesterol acyl transferase is not entirely due to blocking of sulfhydryl groups of the enzyme. The reduction of enzyme activity in vitamin E deficient rats may be due to some direct alteration by oxidation or to some effect secondary to an alteration in phospholipids.

0903 SELENIUM DEFICIENCY AND PROTEIN, RNA AND DNA SYNTHESIS IN RAT PANCREAS AND LIVER. *(E.)* McConnell, K. P.; Hsu, J. M.; Anthony, W. L.; Bieri, J. G. (VA Hosp., Louisville, Ky.). *Proc. Soc. Exp. Biol. Med.* 147(2):575-577, 1974.

Protein, RNA and DNA metabolism were studied in the pancreas and liver of weanling male Sprague-Dawley rats fed a selenium-deficient diet containing vitamin

E (100 mg/kg) for 66 days. Compared to control animals fed a diet supplemented with 0.5 ppm selenium as sodium selenite, selenium-deficient rats had significantly less ($P<0.01$) selenium in the 3 tissues studied (liver, kidney, muscle). Selenium-deficient rats gained less weight (260 gm) than did controls (280 gm). Estimation of the rates of protein, RNA and DNA synthesis by measuring the amount of acid insoluble radioactivity in liver and pancreas following injection of [^{14}C]glycine-2, [^{3}H]uridine-5,6 or [^{3}H]thymidine, showed no significant differences between control and selenium-deficient rats. These results do not lend support for any functional changes in protein, RNA and DNA metabolism in the liver and pancreas of selenium-deficient rats and are not consistent with gross changes in pancreatic enzyme activities as previously reported for selenium-deficient chicks.

0904 IRON DEFICIENCY: IMPAIRED LIVER GROWTH AND DNA SYNTHESIS IN THE RAT. *(E.)* Siimes, M. A.; Dallman, P. R. (Dep. Pediatr., Univ. California, San Francisco). *Br. J. Haematol.* 28(4):453-462, 1974.

The rates of liver growth and DNA synthesis were studied in Sprague-Dawley rats weaned to an iron deficient diet. In the first set of experiments, animals were fed *ad lib* after weaning on day 21 and were sacrificed at various times up to 48 days of age. Decreased liver iron stores and PCV were observed prior to weaning; retarded liver and body weight increase began shortly after weaning. At 48 days of age, the liver iron, PCV, liver weight and body weight were depressed to 89, 64, 36 and 23% below control values, resp. Mean total liver DNA concentration became variably decreased in iron-deficient rats after 33 days of age; however, *in vivo* incorporation of ^{3}H-thymidine into liver DNA and liver thymidine kinase activity were unaffected. Supplementation of the diet of deficient rats with iron dextran for 2 days prior to killing at 48 days increased liver non-heme iron by 601% to values comparable to age-matched controls; PCV increased by 44% and liver and body weights rose 22% and 7%, resp.; no changes occurred in total liver DNA content. In a second set of experiments, rats were given food only twice daily to determine any possible effects of feeding pattern. Total liver DNA and weight gain increased more slowly than in rats fed *ad libitum*. Although ^{3}H-thymidine incorporation was normal over a 24 hr period, incorporation of ^{32}P into liver DNA was decreased in iron-deficient rats. These results indicate that changes in liver DNA synthesis due to iron deficiency occur which are distinct from those seen in starvation and protein-caloric malnutrition.

0905 ALCOHOL, AMINO ACIDS, AND ALBUMIN SYNTHESIS. *(E.)* Rothschild, M. A.; Oratz, M.; Schreiber, S. S. (New York VA Hosp., N.Y.). *Gastroenterology* 67(6):1200-1213, 1974.

The isolated perfused rabbit liver was used to compare the acute effects of alcohol and amino acids on albumin synthesis, polysomal aggregation, and RNA turnover. The perfusion solutions consisted of washed rabbit RBC with a bicarbonate buffer containing 3 g/100 ml rabbit or bovine albumin, 0.08% glucose, test amino acids in a final concentration of 10 mM, and/or alcohol at 220 mg/100 ml. Albumin synthesis was determined isotopically and immunologically and the methods agreed well. [^{3}H]Uridine was added to the perfusion solution to label newly synthesized RNA. Fasting reduced albumin synthesis 50%, and the endoplasmic reticulum-bound polysome was disaggregated. Tryptophan, arginine, ornithine, lysine, phenylalanine, glutamine, alanine, threonine, and proline increased albumin synthesis toward or exceeding the value found in the fed control, and the endoplasmic membrane-bound polysome was reaggregated. Leucine, valine, methionine, and histidine failed to stimulate albumin synthesis or cause reaggregation. The RNA of the bound polysome achieved 60% of the specific activity of that in the free polysome in the fed control preparation, and no change occurred with fasting or after the stimulating amino acids. Total RNA was decreased in fasting and rose after perfusion with the stimulating amino acids. The depression in albumin synthesis, therefore, appears to be related to a decrease in available ribosomes. Alcohol reduced albumin synthesis by 67% and caused marked disaggregation of the endoplasmic membrane-bound polysome. Arginine, trytophan, ornithine, and lysine reversed this depression, and reaggregation with the endoplasmic membrane-bound polysome occurred. Alcohol, however, reduced the bound to free relative specific activity ratio; the addition of stimulating amino acids returned this ratio toward normal. No loss in RNA was noted. Thus, alcohol appears to alter the formation of new polysomes, possibly by impeding the attachment of new labeled ribosomes on to mRNA. The combination of fasting and alcohol, although not reducing albumin synthesis below the level seen with either stress applied alone, prevented the liver from recovery from either stress, in terms of albumin synthesis or polysomal reaggregation.

0906 INCORPORATION OF LABELLED AMINO ACIDS INTO LIVER PROTEIN AFTER ACUTE ETHANOL ADMINISTRATION. *(E.)* Morland, J. (Dep. Pharmacol., Univ. Oslo, Norway). *Biochem. Pharmacol.* 24(3):439-442, 1975.

The effects of acute ethanol administration were studied on incorporation of amino acids into rat hepatic proteins and precursor pools both *in vivo* and using an *in vitro* liver perfusion system. When livers were perfused for 1 to 2.5 hr with solution containing ethanol (0.05-0.30%), incorporation of ^{3}H-valine into protein was unaffected. The proportion of labeled protein remained the same between ethanol-perfused and control livers for at least 1.5 hr after administration of cyclophosphamide, indicating that ethanol also had no effect on protein degradation. The fact that the amount of ^{3}H-valine present in perfused liver 5 min after addition of label was the same for ethanol-treated and controls implied that ethanol had no effect on precursor pool specific activity. Labeled perfusate proteins, however, were reduced from 7.1% to 5.0% of the labeled hepatic proteins after ethanol treatment, suggesting that ethan-

ol either reduced synthesis or release of perfusate proteins. Acute ethanol ingestion (11 g ethanol p.o./kg/day for 4 days) by previously untreated rats resulted in a 183-189% increase in hepatic free leucine and in incorporation of free leucine counts compared to controls, in which sucrose was substituted isocalorically for ethanol in the diet. Incorporation of ^{3}H-leucine into hepatic proteins was not affected by ethanol even after corrections were made for differences in leucine precursor pool specific activity. Acute ethanol administration to rats given ethanol for 4 weeks increased free leucine concentration to 153% and free leucine radioactivity to 263% of the control values and caused a 55% reduction in ^{3}H-leucine incorporation into hepatic protein. These results combined with previously published reports in which ethanol reduced hepatic uptake of 2 other amino acids suggest that ethanol exerts selective effects on the various amino acid membrane transport systems.

0907 EFFECT OF ETHANOL ON THE METABOLISM OF ALANINE, GLUTAMIC ACID, AND PROLINE IN RAT LIVER. *(E.)* Hakkinen, H. M.; Kulonen, E. (Res. Lab. State Alcohol Monop., Helsinki, Finland). *Biochem. Pharmacol.* 24(2):199-204, 1974.

Alanine, glutamic acid, and proline metabolism were studied in liver homogenates from male Wistar rats given ethanol (550 mg/100 g, 60 min) by stomach tube and in normal, rat liver slices incubated in ethanol (20 mM, 2 hr). Net formation of alanine in ethanol-treated liver homogenates as determined from electrophoretic assay was decreased to 30.8% of control. Enzymatic determinations showed that synthesis of glutamic acid from α-ketoglutaric acid was increased by about 33% in both ethanol-treated liver homogenates and slices. The inability of glutamic acid to accumulate in normal liver homogenates incubated with ethanol *in vitro* indicates that an intact cellular process is necessary for this effect. Ethanol also stimulated free proline accumulation by 50-60% both in liver slices and in homogenates from *in vivo* treated rats at all glutamine concentrations used. The ratio of proline to glutamic acid was not, however, changed. Addition of lactic acid to the homogenate incubation mixture stimulated proline formation from glutamic acid by 37-100%. Thus, increased proline availability to hepatic fibroblasts secondary to ethanol effect may be one mechanism by which connective tissue is activated in liver to develop alcoholic cirrhosis.

0908 CHARACTERISTICS OF ETHANOL AND ACETALDEHYDE OXIDATION ON FLAVIN AND PYRIDINE NUCLEOTIDE FLUORESCENCE CHANGES IN PERFUSED RAT LIVER. *(E.)* Lindros, K. O.; Oshino, N.; Parrilla, R.; Williamson, J. R. (Johnson Res. Found., Univ. Pennsylvania, Philadelphia). *J. Biol. Chem.* 249(24):7956-7963, 1974.

The percentage decomposition of the catalase-H_2O_2 intermediate by methanol and ethanol was determined from absorbance changes (660-640 nm) and used to calculate the intracellular ethanol concentration in the lobes of *in vitro* perfused male albino rat livers. Values obtained were similar to those measured in the effluent fluid from the liver. Using this technique, a value of 0.09 mM for the apparent K_m of alcohol dehydrogenase *in situ* for ethanol and a V_{max} value of 1.25 μM/min/g wet weight of liver were calculated. The hepatic ethanol concentration giving half-maximal reduction of the pyridine nucleotides as determined from pyridine nucleotide fluorescence changes (366-nm excitation, 460-nm emission) was 0.07 mM. When arterial fluid ethanol concentrations were used, the apparent K_m for ethanol was about 0.4 mM. The apparent K_m values for ethanol measured *in situ* are 5- to 20-fold lower than values measured with isolated rat liver alcohol dehydrogenase, indicating that ethanol metabolism by the liver is not directly limited by alcohol dehydrogenase activity but rather by the rate of reoxidation of cytosolic NADH. At the low rates of hydrogen peroxide production observed in substrate-free perfused livers, rates of ethanol oxidation *via* alcohol dehydrogenase calculated from the different effectiveness of ethanol and methanol in decomposing the catalase-H_2O_2 intermediate were similar to rates of ethanol uptake measured directly from arteriovenous concentration differences. Changes of flavin and pyridine nucleotide fluorescence as measured by surface fluorometry were compared with changes in the ratios of lactate to pyruvate and β-hydroxybutyrate to acetoacetate in the effluent fluid perfusing the liver. Titrations with octanoate to reduce the pyridine nucleotides in the mitochondrial compartment showed that the oxidation-reduction state of the fluorescent flavoproteins was in rapid equilibrium with the ratio of β-hydroxybutyrate to acetoacetate. A comparison of pyridine nucleotide oxidation-reduction changes during ethanol infusion, and titrations with acetaldehyde in the absence and presence of 4-methylpyrazole (an inhibitor of alcohol dehydrogenase), showed that at acetaldehyde concentrations above 0.2 to 0.4 mM a dismutation between alcohol and acetaldehyde dehydrogenase occurred in the cytosolic compartment. At lower acetaldehyde concentrations commensurate with those occurring during ethanol oxidation, the evidence indicates that acetaldehyde is normally oxidized in the mitochondrial compartment.

0909 HEPATIC TRYPTOPHAN OXYGENASE ACTIVITY AS A MARKER OF CHANGES IN PROTEIN METABOLISM DURING CHRONIC ETHANOL TREATMENT. *(E.)* Morland, J. (Inst. Pharmacol., Univ. Oslo, Norway). *Acta Pharmacol. Toxicol. (Kbn.)* 35(2):155-168, 1974.

Hepatic tryptophan oxygenase activity was studied in blood samples from male Wistar rats fed a diet containing ethanol (22, 30 or 38% of the total caloric intake). Controls received a diet containing isocaloric amounts of sucrose instead of ethanol. Rats receiving ethanol as 30 and 38% of the calories showed significant reductions in tryptophan oxygenase activity after 7 and 21 days, resp., which lasted for the remainder of the experiment (total of 50 days). Rats receiving ethanol (22%) showed no decrease. Hepatic triglyceride content, plasma lipids, plasma enzyme activities (alkaline phosphatase, SGOT and SGPT) and hepatic histology were only slightly or transiently affected. *In vitro* liver perfusion experiments using organs from rats treated with ethanol for 7 or 21 days showed a 30% reduction in dexamethasone-induced tryptophan oxygenase and a 20% decrease in tyro-

sine transaminase activity. Incorporation of isotopically-labeled amino acids into perfused livers of ethanol-treated rats (21 days) was reduced by 20% after 1 hr although the specific activity of proteins as well as acid-soluble counts were unchanged. Ethanol treated rats also showed a slight insignificant reduction in liver and plasma proteins. Protein electrophoresis revealed no qualitative changes in ethanol-treated animals. It was thus concluded that tryptophan oxygenase activity is most probably an indicator of the synthesis of this enzyme as well as of other proteins sensitive to chronic ethanol administration.

0910 LIPOPROTEIN LIPASE ACTIVITY OVER ACUTE ALCOHOLIC POISONING. *(E.)* Mitkov, D. (I.P. Pavlov Higher Med. Inst. Plovdiv, Bulgaria). *Folia Med. (Plovdiv)* 15(3):135-140, 1973.

Serum lipoprotein lipase activity was studied in 80 fasted white rats following gastric intubation of a 50% ethanol solution (9.3 gm/kg) and i.v. administration of heparin (50 U/100 gm) 16-18 hr later. Total plasma lipid levels were greatest 16-18 hr after ethanol intubation (610 mg% compared to 354 mg% in controls). Serum lipoprotein lipase activity was also greatest at this time, showing increased activity in response to i.v. heparin administration when compared to heparin-treated controls. Since previous studies have shown free heparin levels to be decreased 16-18 hr after acute ethanol ingestion in rats, these results indicate that acute ethanol ingestion is most probably associated with lowered free plasma heparin which inhibits secretion of lipoprotein lipase into the blood resulting in delayed hydrolysis of triglycerides. Thus ethanol-induced hyperlipemia is most likely of the retention type.

0911 RADIOSCINTIGRAPHIC STUDIES OF ^{11}C DISTRIBUTION IN CATS GIVEN 1-^{11}C-ETHANOL. *(E.)* DeGrazia, J. A.; Rodden, A. F.; Teresi, J. D.; Busick, D. D.; Walz, D. R. (Stanford Univ Sch. Med., Calif.). *J. Nucl. Med.* 16(1):73-76, 1974.

The distribution of ^{11}C-isotope following i.v. injection of ^{11}C-ethanol into 8 cats was studied using gamma-ray scintigraphy. Over 50% of the ^{11}C-isotope was present in the liver and lungs of cats shortly after being given a high specific activity dose. Peripheral tissues equilibrated more slowly. Liver ^{11}C-isotope content was relatively decreased and lung activity was correspondingly increased in animals given a low specific activity ^{11}C-ethanol dose. These changes most likely reflect changes in the rate of hepatic ethanol metabolism. The overall tissue content of ^{11}C-isotope in these studies was directly proportional to the amount of ^{11}C-ethanol administered. Thus, ^{11}C-labeled compounds may possibly be used to obtain useful *in vivo* information by gamma-ray scintigraphy, particularly with respect to the metabolic functions of various organs.

0912 ACUTE ALCOHOLIC FATTY LIVER -- METABOLISM OR STRESS. *(E.)* Kalant, H.; Khanna, J. M.; Seymour, F.; Loth, J. (Dep. Pharmacol., Univ. Toronto, Canada). *Biochem. Pharmacol.* 24(3):431-434, 1974.

Hepatic triglyceride accumulation was studied in male Wistar rats following ethanol administration. Animals which received a single, intoxicating dose of ethanol (4.8 g/kg) by stomach tube had hepatic triglyceride levels (13.68 mg/g liver) which were twice those of the controls (6.71 mg/g) which received a single dose of glucose. When the same total dose of ethanol was given in 4 equally divided, nonintoxicating portions 3 hr apart, hepatic triglycerides were elevated (10.24 mg/g) but not to the extent that they were following the single dose. Peak blood ethanol level following the single dose was 400 mg/180 ml after 2-4 hr and during administration of the divided doses was 122 mg% at 10 hr. The 2 curves, however, were superimposable after the 10th hr, suggesting that the microsomal oxidizing system was not contributing to ethanol disappearance from the blood. The fact that the differences in hepatic triglycerides between the 2 feeding schedules were not seen in animals used the day following arrival to the laboratory was interpreted to indicate that high blood adrenalin due to stress was present in this group. It is concluded that, although ethanol metabolism is primarily responsible for triglyceride buildup, intoxication and the direct effect of ethanol *per se* also contributes significantly.

0913 VIRAL POTENTIATION OF CHEMICAL TOXINS IN THE EXPERIMENTAL SYNDROME OF HYPOGLYCEMIA, ENCEPHALOPATHY, AND VISCERAL FATTY DEGENERATION. *(E.)* Colon, A. R.; Ledesma, F.; Pardo, V.; Sandberg, D. H. (Univ. Miami Sch. Med., Miami, Fla.). *Am. J. Dig. Dis.* 19(12):1091-1101, 1974.

In an attempt to further elucidate the pathogenesis of the syndrome of hypoglycemia, encephalopathy and fatty degeneration of the liver, the effects of various hepatotoxins, including 4-pentenoic acid, aflatoxin B, cuprizone, polychlorinated biphenyl and lindane, with or without concomitant mengo virus infection, were studied on the livers of fed and fasted Sprague-Dawley rats. The most striking and consistent changes were observed in virus-infected rats given 4-pentenoic acid (100 mg/kg/day i.p., i.m. or i.v.). Medullary vacuolization and pericellular clear spaces were common in these animals. Livers demonstrated fine vacuolization and hepatocyte glycogen depletion was confirmed cytochemically. Electron microscopy showed, in addition to those changes already noted, the presence of prominent electron-dense granules within swollen mitochondria. These granules measured from 0.1 to 1.0 μm and showed a finely fibrillar structure. Although their origin is obscure, they were observed only in rats exposed to pentenoic acid and mengo virus. Pentenoic acid treated, mengo virus infected animals were frequently hypoglycemic, and hepatic triglyceride steatosis was marked. The virus-accentuated hepatic steatosis observed in these studies supports the hypothesis that the syndrome of hypoglycemia, encephalopathy and fatty visceral degeneration may result from potentiation of an hepatic mitochondrial toxin by viral infection.

0914 FATTY LIVER PRODUCED BY HYPERALIMENTATION OF RATS. *(E.)* Chang, S.; Silvis, S. E. (V.A. Hosp., Minneapolis, Minn.). *Am. J. Gastroenterol.* 62(5):410-418, 1974.

Fatty liver formation was studied in restrained male Sprague-Dawley rats infused with a hypertonic dextrose solution, with or without amino acid supplementation, through a superior vena cava catheter delivering 130 to 341 cal/kg/day. Infusion for 4 days of hypertonic dextrose at a high caloric level (>300 cal/kg/day) resulted in gross fatty infiltration of the livers of 8 of 9 rats, which was not prevented by addition of essential amino acids at levels exceeding the minimal daily oral requirement. Addition of a fibrin hydrolysate showed some sparing effect in animals infused at a rate of 250 cal/kg/day but had no effect in animals receiving 315 cal/kg/day. Total liver lipid content was measured in 60 rats and was significantly increased in animals infused with 209 cal/kg/day. Total hepatic lipid levels, however, had no predictive value with respect to the degree of fatty infiltration. Although all animals infused with dextrose had increased liver glycogen, total levels were < 600 mg and accounted for only a small fraction of liver weight gain which was directly related to caloric intake (r=0.68). Although some rats were in negative nitrogen balance, fatty liver was observed even in the presence of a positive nitrogen balance. The induction of fatty liver under these conditions was thus felt to be primarily a result of high caloric intake although possible influences of restraint and negative nitrogen balance could not be totally ruled out.

0915 STUDIES ON THE PATHOGENESIS OF EXPERIMENTAL CHRONIC ACTIVE HEPATITIS IN RABBITS: I. INDUCTION OF THE DISEASE AND PROTECTIVE EFFECT OF ALLOGENEIC LIVER SPECIFIC PROTEINS. *(E.)* Meyer Zum Buschenfelde, K. H.; Hopf, U. (2nd Dep. Intern. Med., Univ. Mainz, Germany). *Br. J. Exp. Pathol.* 55(5): 498-508, 1974.

The protective effect of allogeneic liver specific proteins on the development of histologically demonstrable liver lesions was studied in 65 white New Zealand rabbits which underwent long-term i.p. immunization for periods from 18 to 143 weeks with human liver specific proteins (25 mg i.p. each injection) plus complete Freund's adjuvant. Immunization with human protein produced a chronic active hepatitis or liver cirrhosis in all animals. Autoimmunity as demonstrated by skin testing was induced by human liver protein against the human liver antigens and persisted following subsequent immunization with rabbit liver protein (5-25 mg each injection). Combined administration of human and rabbit liver protein (native and aggregate-free, resp.) caused histologically documented liver lesions in only 1 of 8 (13%) animals. Skin testing with rabbit liver protein was negative in 6 and weakly positive in the other 2 rats after 14 weeks. The humoral immune reactions in these animals were the same as those observed in human liver protein immunized rats: a decreasing titer against rabbit liver protein. Rats receiving only complete Freund's adjuvant (1-2 ml each infection) neither developed positive antibody titers nor developed chronic active hepatitis or liver lesions. The pathogenesis of experimental chronic active hepatitis under these conditions thus appears to be mediated by a cellular rather than humoral toxicity.

0916 HEPARINOIDS -- A NEW INHIBITOR OF EXPERIMENTAL LIVER NECROSIS IN RATS. *(E.)* Bartsokas, S. K.; Papadimitriou, D. G.; Papacharalampous, N. X.; Katapoti, Z.; Kannas, D. (Med. Fac., Athens Univ., Greece). *Acta Hepatogastroenterol. (Stuttg.)* 21(4):253-260, 1974.

To determine whether the heparinoid sodium pentosan polysulfate directly affects thioacetamide-induced liver cell necrosis and whether it induces hypothermia, adult Wistar rats were poisoned by a 2% aqueous solution of thioacetamide (200 mg/kg i.p.). Some groups of rats (32-48/group) were given 10 or 20 mg of the heparinoid immediately after poisoning and 6 hr later, and others were given additional doses 12 and 18 hr later. Controls were either untreated or injected i.p. with distilled water at the same dosages and times as the heparinoids. Rats were sacrificed from 24-96 hr after poisoning. Liver tissues were stained with hematoxylin and eosin and observed by light microscopy. No differences were found between the lesions found in the untreated group and the group given the distilled water. Groups that had been given lower total dosages, either through amount injected or number of injections, showed greater necrosis than those given higher total dosages, including those sacrificed at 72 and 96 hours. No lowering of rectal temperature was found in the treated groups. It is concluded that sodium pentosan polysulfate protects the liver against thioacetamide toxicity by directly preventing liver cell necrosis.

0917 THE EFFECT OF THE HYPOCHOLESTEROLEMIC DRUG CLOFIBRATE ON LIVER MITOCHONDRIAL BIOGENESIS: A ROLE FOR NEUTRAL MITOCHONDRIAL PROTEASES. *(E.)* Gear, A. R. L.; Albert, A. D.; Bednarek, J. M. (Dep. Biochem., Univ. Virginia, Charlottesville). *J. Biol. Chem.* 249(20):6495-6504, 1974.

The action of clofibrate on liver mitochondrial levels was investigated to confirm the hypothesis that the drug's hypolipemic action is correlated with the increased mitochondrial levels. Male albino Sprague-Dawley rats were given the ethyl ester of clofibrate in a concentration of 0.3% (w/w) in their diet. Mitochondria were isolated by differential centrifugation, and the heavy and light fractions combined. Mitochondrial neutral protease was assayed at intervals from 10 to 20 days after initiation of treatment. Clofibrate was found to produce a 100% increase in hepatic mitochondrial content within 10 days. To test whether a change in yield was responsible, total cytochrome c oxidase activity in the nuclear, mitochondrial, and supernatant fractions were assayed and found to be the same in experimental and control animals. At 6 days on clofibrate, there was a significant increase in respiratory control. A decrease in the outer mitochondrial enzymes, such as during massive mitochondrial biogenesis, was noted. However,

no significant increases in mitochondrial DNA or RNA were found. No differences between experimental animals and controls were found in half-life studies of the mitochondria, using guanidinio-^{14}C-labeled arginine. However, the synthesis constant was determined by use of ^{35}S-labeled methionine, sacrificing rats after 10, 20, 30, and 60 min. At the shortest times, stimulation of protein synthesis was found. Mitochondrial neutral protease activity was found to be significantly inhibited by monitoring the pattern of phosphate inhibition. The *in vitro* effect of the drug on protease activity was also determined, using an aqueous solution of the sodium salt. No significant inhibition was found. The possibility of lysosomal contamination affecting the *in vivo* results was also eliminated. It is concluded that clofibrate inhibits mitochondrial neutral protease activity and raises the level of newly synthesized protein for incorporation into mitochondria.

0918 BIOCHEMICAL STUDIES ON THE MECHANISM OF ACTION OF LIVER POISONS. III. DEPLETION OF LIVER GLUTATHIONE IN ETHIONINE POISONING. *(E.)* Glaser, G.; Mager, J. (Hadassah Med. Sch., Jerusalem, Israel). *Biochim. Biophys. Acta* 372(1):237-244, 1974.

The mechanism of ethionine poisoning in albino rats was studied with regard to the role of altered glutathione metabolism. Protein synthesis was measured by ^{14}C labeled phenylalanine using a liquid scintillation spectrometer. Oxidized glutathione content was measured by its reduction by glucose-6-phosphate. ATP was determined by the luciferin-luciferase procedure. It was found that in female rats, reduced glutathione liver content and protein synthesizing capacity (measured in cell-free liver preparation) fell rapidly to 40-50% of normal values within 3 hr of ethionine administration. Male and adrenalectomized female rats showed no such decline until after 3 hr. Both sexes showed an equal degree of ATP depletion. It was assumed that the early reduced glutathione decline in the females was due to excessive formation of the oxidized form, which was then immediately destroyed. This was supported by the finding that whole liver homogenates destroyed oxidized glutathione rapidly. There was no difference between normal and ethionine-treated female liver homogenates in capacity to oxidize glutathione, and no difference observed when liver homogenates were fortified with glucose-6-phosphate and NADP. It is suggested that ethionine did not impair the NADPH-linked glutathione reductase. When glucose-6-phosphate was omitted, glutathione reduction was curtailed in treated females, but not males or adrenalectomized females. It is concluded that reduced glutathione depletion by ethionine is not directly related to ATP depletion. It is due to improved glutathione reduction capacity resulting from inadequate substrate for NADPH generation.

0919 BIOCHEMICAL INDICES OF TETRACYCLINE HEPATIC INJURY IN RATS. *(E.)* Shauer, B. A.; Lukacs, L.; Zimmerman, H. J. (VA Hosp., Washington, D.C.). *Proc. Soc. Exp. Biol. Med.* 147(3):868-872, 1974.

Hepatic morphologic and biochemical injury were studied in female Wistar rats following i.p. instillation of tetracycline (100-300 mg/kg). Small droplet fatty infiltration was noted by light microscopy in the livers of all experimental animals by 24 hr after tetracycline administration and reached a maximum extent after 48 hr. These changes were corrected with a 3- to 4-fold increase in SGOT levels after 24 hr compared to controls. A dose of 50 mg/kg produced less than a 2-fold increase in SGOT activity after 24 hr and had no morphologic effect. Changes in SGPT activity were also observed in tetracycline-treated animals (200 mg/kg) but were less sensitive than SGOT levels in reflecting hepatic injury. Serum levels of glutamic dehydrogenase, acid and alkaline phosphatase, β-glucuronidase and bilirubin remained unchanged following tetracycline instillation. The hepatic morphologic and SGOT and SGPT changes following tetracycline administration differed in quality and quantity from those previously observed for nutritional fatty metamorphosis (little or no SGOT and SGPT elevation) and carbon tetrachloride-induced liver necrosis (>100-fold SGOT increase starting within 3 hr).

0920 STUDIES ON THE EFFECTS OF TRICAPRYLIN ON GLUCONEOGENESIS AND KETOGENESIS IN ISOLATED PERFUSED LIVER. *(E.)* Ingebretsen, Jr., W. R.; Wagle, S. R. (Indiana Univ. Sch. Med., Indianapolis). *Proc. Soc. Exp. Biol. Med.* 147(2):578-580, 1974.

The effects of gluconeogenic substrates on ketogenesis and the effects of tricaprylin on gluconeogenesis were studied in perfused livers from 18-24 hr fasted male Cox rats. Addition of 10 mM/galactose, lactate, pyruvate or alanine to the perfusion medium suppressed endogenous ketone body formation by 50-75%. Tricaprylin (1 mM) caused a 20-fold increase in ketogenesis in the absence of carbohydrate. Alanine, pyruvate and galactose had no effect on the increase in ketogenesis in the presence of tricaprylin; lactate, however, reduced the increase in ketone body formation by tricaprylin by about 60%. Lactate had no effect on octanoate-induced stimulation of ketogenesis in this system. Studies conducted on *in vitro* gluconeogenesis showed that tricaprylin stimulated glucose formation by about 2-fold in the presence of 10 mM lactate, alanine or galactose but not pyruvate. Octanoate alone had no effect on glucose production from lactate. It was, however, equally as effective as tricaprylin when the two were present in equimolar quantities, indicating that the increase in glucose synthesis is due to the octanoate moiety of tricaprylin. The rate of ketogenesis did not correlate with the rate of gluconeogenesis under these conditions. These results are in agreement with earlier studies which indicate that fatty acid oxidation is needed for high rates but not for low rates of glucose production. Thus, fatty acid oxidation may regulate gluconeogenesis under conditions of increased availability (eg, hormonal stimulation, starvation or diabetes).

0921 DEPRESSION OF HEPATIC GLUCONEOGENESIS BY ACUTE LEAD ADMINISTRATION. *(E.)* Cornell, R. P.; Filkins, J. P. (Dep. Physiol., Loyola Univ.,

Maywood, Ill.). *Proc. Soc. Exp. Biol. Med.* 147(2): 371-376, 1974.

The effects of acute i.v. lead administration on hepatic gluconeogenesis were studied in male Holtzman rats. The rats were injected with either lead acetate or sodium acetate (5 mg) and were fasted overnight. Hepatic gluconeogenesis was estimated the following morning following i.v. injection of unlabeled and/or isotopically labeled L-alanine or sodium pyruvate. In addition, gluconeogenesis was similarly assessed in preparations of hepatocytes from lead-injected, fasted rats. Compared to sodium acetate-injected controls, lead acetate-injected rats showed both an insignificant hyperglycemic response following alanine or pyruvate injection and an insignificant rate of incorporation of isotopically labeled substrates into blood glucose. Isolated control hepatocytes synthesized glucose from alanine, lactate, pyruvate, oxaloacetate, glycerol and fructose, whereas hepatocytes from lead-intoxicated rats showed comparable rates of gluconeogenesis only from glycerol. Gluconeogenesis from all substrates requiring obligatory mitochondrial processing was depressed to the greatest extent in lead-treated animals. Incorporation of ^{14}C-alanine into carbon dioxide was also depressed in lead-treated hepatocytes. These results suggest that lead exerts its effects at the level of the mitochondrion and that defects in hepatic glucoregulation may play a role in the toxicity of acute lead poisoning.

0922 A STUDY OF THE RELATIONSHIPS BETWEEN CARBON TETRACHLORIDE-INDUCED LIPID PEROXIDATION AND LIVER DAMAGE IN RATS PRETREATED WITH VITAMIN E. *(E.)* Benedetti, A.; Ferrali, M.; Chieli, E.; Comporti, M. (Inst. Gen. Pathol., Univ. Pisa, Italy). *Chem. Biol. Interact.* 9(2):117-134, 1974.

The relationships between the peroxidation of microsomal lipids and the early liver damage have been investigated in rats pretreated with progressively higher doses of α-tocopherol (vitamin E) and intoxicated with various amounts of carbon tetrachloride. Pretreatment of rats orally with vitamin E at 25 mg/100 g had minor effects on both the peroxidation of microsomal lipids (as measured by diene conjugation) and the liver triglyceride accumulation in rats poisoned with CCl_4 at 250 µl/100 g by gastric intubation. However, a decrease of the peroxidative reaction and of the liver steatosis occurred when the rats were pretreated with progressively higher doses of vitamin E. A close correlation existed between the two phenomena when the intoxication was accomplished with CCl_4 at 250 µl/100 g. Also, the microsomal concentration of α-tocopherol was strictly correlated to both the decrease of microsomal lipoperoxidation and the decrease of liver triglyceride accumulation. The CCl_4-induced impairment of microsomal glucose-6-phosphatase and the incorporation of ^{14}C from $^{14}CCl_4$ into liver microsomal lipids were not affected by vitamin E pretreatment. The extent of microsomal lipoperoxidation was not correlated to the liver triglyceride accumulation when vitamin E-pretreated rats were given CCl_4 at 25 or 2.5 µl/100 g. However, a correlation between lipoperoxidation and liver steatosis occurred when non-pretreated rats were challenged with the three different doses of the halogenated hydrocarbon. Thus, the effect of α-tocopherol in decreasing the extent of microsomal lipid peroxidation in rats given CCl_4 (250 µl/100 g) can be explained by an antioxidant activity *in vivo* against the pro-oxidant effect of CCl_4.

0923 INDUCTION OF GRANULOMATOUS HEPATITIS IN MICE INFECTED WITH GROUP A STREPTOCOCCI AND TREATED WITH PENICILLIN. *(E.)* Spanel, R.; Galle, J.; Heymer, B.; Haferkamp, O.; Schmidt, W. C. (Dept. Pathol., Univ. Ulm, Germany). *Virchows Arch.* [*Pathol. Anat.*] 363(4):323-332, 1974.

The development of granulomatous hepatitis was studied in 115 inbred CBA/H male and female mice following infection with live group A streptococci (T22/76/2, type 22, 0.5-3.0 mg i.v.) and immediate penicillin G treatment (50,000 U s.c. at 1, 16 and 40 hr). Granulomatous foci which appeared in the livers of infected, treated animals after 8-12 days closely resembled those induced by heat-killed group A streptococci. The only difference between these 2 groups was in the initial phase of induction: in addition to macrophages and lymphocytes, livers of mice injected with live bacteria also contained polymorphonuclear granulocytes. The lesions resembled the entity known as "non-specific granulomatous hepatitis" seen in humans, some of whom had been treated with antibiotics for a recent pyogenic coccal infection. Both the number and size of hepatic granulomas were, in both groups, correlated with the amount of streptococcal particles and antigens in the livers as detected by electron microscopy and immunofluorescence. No other evidence of chronic liver inflammatory disease was seen. The spleen, however, showed a non-specific inflammatory reaction most pronounced between the 4th and 12th day. Mice injected only with live group A streptococci or with penicillin or with latex particles did not develop liver granulomas. There was no evidence that penicillin treatment accelerated elimination of streptococcal components from tissue.

0924 EXPERIMENTAL ACUTE ENCEPHALOPATHY: COMPARISON OF THE ELECTROENCEPHALOGRAPHIC CHANGES IN THE LIVERLESS AND IN THE EVISCERATED RAT. *(E.)* Degos, F.; Degos, J. D.; Bourdiau, D.; Peignoux, M.; Prandi, D.; Roche-Sicot, J.; Sicot, C.; Rueff, B.; Benhamou, J. P. (INSERM, Beaujon Hosp., Clichy, France). *Clin. Sci. Mol. Med.* 47(6):599-608, 1974.

EEG changes in hepatectomized rats were compared with those in eviscerated rats to rule out the possibility that hepatic encephalopathy develops from intestinal release of substances such as ammonia. Male Charles River rats with vena caval ligation and end-to-side portacaval shunts were either hepatectomized (8) or eviscerated (24). In the eviscerated rats both the liver and the other splanchnic viscera were removed. Rats in the sham-operated group (8) were subjected to 20 min laparotomies, and those in the normal group (20) were untreated. Rectal temperatures of both hepatectomized and eviscerated rats and of one group (11) of sham-operated rats were maintained at 26-28 C. In the control rats, 4 states of wakefulness and

sleep were found: active wakefulness, quiet wakefulness, slow sleep, and paradoxical sleep. Both normal and refrigerated sham-operated rats were in a continuous state of active or quiet wakefulness with occasional slow sleep EEG patterns. Both the eviscerated and hepatectomized rats showed similar patterns of brain disorders: motionlessness, lack of stimuli response, and intermediate voltage slow waves in the EEG from the first 1-2 1/2 hr; sudden arousal, restlessness and alternating active and quiet wakefulness EEG patterns which slowly changed to motionlessness and slow sleep patterns occurred from 2-11 hr; motionlessness and slow sleep EEG's with deteriorating stimulus responses beginning from 4-13 hr and lasting 5-25 hr. Eventually the EEG tracings became flat and stimulus response was lost. These changes were not related to hypoglycemia, since blood glucose levels were maintained above 6.11 mM/liter; nor were they related to hypothermia, since they were not observed in the refrigerated sham-operated rats. They were also unrelated to low blood pressure. The values for plasma ammonia were significantly lower in the eviscerated rats. The results suggest that encephalopathy induced by hepatectomy and that induced by evisceration are of the same nature, and that the increase in plasma ammonia (or any other neurotoxic substance released from the intestine) has little role in the pathogenesis of hepatic encephalopathy.

0925 METABOLISM OF STEROID AND AMINO ACID MOIETIES OF CONJUGATED BILE ACIDS IN MAN: IV. DESCRIPTION AND VALIDATION OF MULTICOMPARTMENT MODEL. *(E.)* Hoffman, N. E.; Hofmann, A. F. (Mayo Clin., Rochester, Minn.). *Gastroenterology* 67(5):887-897, 1974.

A kinetic multicompartment model has been described for the enterohepatic circulation of conjugated bile acids in man which encompasses bile acid synthesis, amino acid conjugation, bacterial deconjugation, hepatic reconjugation and bacterial dehydroxylation. Mean data from 3 separate groups of healthy subjects were used to assign relative magnitudes to the rate constants. Use of the model to predict the fate of ring labeled bile acids showed good agreement with observed values. The potential errors of the commonly used techniques for estimating bile acid kinetics have been compared and the validity of the Lindstedt method has been confirmed (administration of tracer as free bile acid with specific activity measurements made on combined conjugates of that acid). However, a large error in calculated values for bile acid pool size and input may occur if cholyltaurine is used as tracer and kinetics are calculated only from its specific activity decay curve. The equations necessary to describe biliary bile acid composition for the steroid and the amino acid moiety are also presented, and it is shown that equilibrium composition is the result of hepatic and intestinal events. For both the steroid and amino acid moiety, biliary bile acid composition *per se* gives little information on output rates. This model is applicable to animals other than man with appropriate modifications.

0926 ARE THERE PROLIFERATIVE COMPARTMENTS IN THE GALLBLADDER? *(E.)* Scott, A. J. (Auckland Univ. Sch. Med., New Zealand). *Gastroenterology* 67(6):1231-1237, 1974.

The immediate pattern of nuclear labeling with ^{3}H-thymidine was investigated to determine if proliferative compartments exist in the guinea pig gallbladder. In the first study, 6 guinea pigs were given ^{3}H-thymidine, 0.1 μCi/g i.p. and killed 6 hr later. In the second study, the common bile duct was ligated in 4 guinea pigs 24 hr before they were given ^{3}H-thymidine, 0.1 μCi/g i.p. They were killed 6 hr later. In the normal gallbladder, nuclear labeling was sparse and without detectable relation to any anatomic feature. In the cystic duct, the surface epithelium, the ductules, and the gland acini all showed occasional early labeling. After common duct ligation, label was strongly and uniformly incorporated in cell nuclei in all structures, except the glands of the gallbladder itself. It is concluded that there are no proliferative compartments in the gallbladder of the guinea pig.

0927 TAURINE SYNTHESIS, CONCENTRATION, AND BILE SALT CONJUGATION IN RAT, GUINEA PIG, AND RABBIT. *(E.)* Spaeth, D. G.; Schneider, D. L. (Dep. Nutr. Res., Mead Johnson Res. Cent., Evansville, Indiana). *Proc. Soc. Exp. Biol. Med.* 147(3):855-858, 1974.

The precentage of bile salts conjugated with taurine in bile, as well as taurine concentration and cysteinesulfinic acid and cysteic acid decarboxylase activities were studied in liver, kidney, brain, lung and heart tissues from adult male Wistar rats, guinea-pigs and rabbits. Taurine conjugates constituted 87% of total conjugated bile salts in the rat, 20% in the guinea-pig and 6% in the rabbit. In all 3 species, an overall correlation was observed between the percentage of taurine conjugates and activities of cysteinesulfinic and cysteic acid decarboxylase. The percentage of taurine conjugates was also correlated with the amount of taurine synthesized per g liver but showed no relation to liver taurine concentration. Taurine synthetic capacity for the various tissues was: kidney> brain≥lung>heart with liver capacity being variable. In general, tissue taurine concentration varied inversely with tissue decarboxylase activity. Taurine concentration in all 3 species was 7-fold greater for heart than for any other tissue studied.

0928 GASTRO-INTESTINAL HORMONES AND BILE SECRETION IN THE PERFUSED PIG LIVER: THE EFFECTS OF SECRETIN, CHOLECYSTOKININ AND PENTAGASTRIN. *(E.)* Jablonski, P.; Sali, A.; Watts, J. M. (Prince Henry's Hosp., Melbourne, Australia). *Aust. NZ J. Surg.* 44(2):173-178, 1974.

Perfused livers obtained from pigs (20-30 kg) were infused with solutions of secretin, cholecystokinin and pentagastrin *via* the vena caval line for a period of 90 min. During all experiments, the enterohepatic circulation of bile acids was mimicked by the infusion of 2% sodium taurocholate into the vena caval line at the rate of 11 μM/min. Pentagastrin and cholecystokinin-pancreozymin were dis-

solved in Tis-u-sol infused at rates of 50 µg and 76 Ivy dog U/hr, resp. Secretin was dissolved in Tis-u-sol containing 0.001 M HCl and infused at a rate of 0.75 U/hr. Bile secretion rate was estimated by weighing timed bile samples. An increase in the bile secretion rate and bicarbonate output was found during secretin infusion. Simultaneous infusion of either cholecystokinin or pentagastrin with secretin augmented this increase to a level greater than the sum of the single hormone responses. Secretin produced a fall in bile-salt output, but neither cholecystokinin nor pentagastrin had any such effect. When either cholecystokinin or pentagastrin was infused together with secretin, however, there was an increase in bile salt output for 30 min, followed by maintenance of output at base-line values. The addition of hormones with the same C-terminal pentapeptide (pentagastrin and cholecystokinin) thus reversed the inhibitory action of secretin on hepatic bile-salt excretion. It is concluded that all 3 hormones influence the handling of bile salts by the hepatocyte.

0929 EFFECT OF CALCITONIN ON THE FORMATION, COMPOSITION, AND ENZYMATIC ACTIVITY OF THE HEPATIC BILE IN GUINEA PIGS. *(E.)* Tarnawski, A.; Bogdal, J.; Dura, K.; Marszalek, Z.; Jedrychowski, A. (Med. Acad., Krakow, Poland). *Gut* 15(9):703-705, 1974.

Ten guinea pigs (average weight 343.5 g) were given calcitonin i.m. in a single dose of 20 MRC U/animal in 0.25 ml gelatin diluent. A control group of 10 animals received the gelatin diluent only. The bile was collected *via* a cannula in the common bile duct for 180 min after the injection of calcitonin or diluent. After this period, blood was obtained by cardiac puncture. The animals were killed and specimens of liver tissue excised. The total volume of bile secreted within 180 min was 2.42 ml/100 g for the controls and 2.54 ml/100 g after calcitonin; thus, calcitonin had practically no effect on bile flow. It also did not effect bile pH, electrolyte composition including calcium, nor the concentration of protein, glucose, cholesterol, nonprotein nitrogen, and bile acids, nor the activities of alanine aminotransferase and alkaline phosphatase in the hepatic bile. Calcitonin decreased the bilirubin concentration and increased the activity of gamma glutamyl transpeptidase in the hepatic bile and increased the calcium content in the liver. The decrease in bilirubin concentration suggests that calcitonin selectively inhibits the transport of bilirubin into the bile, probably by inhibition of enzymes involved in this transport. The increase of gamma glutamyl transpeptidase activity may be the result of induction of this enzyme in the liver or its enhanced excretion by hepatocytes into the bile or both.

0930 BILE ACIDS: XLIV. QUANTITATION OF BILE ACIDS FROM THE BILE FISTULA RAT GIVEN [4-^{14}C]CHOLESTEROL. *(E.)* Siegfried, C. M.; Doisy, Jr., E. A.; Elliott, W. H. (St. Louis Univ. Sch. Med., Mo.). *Biochim. Biophys. Acta* 380(1):66-75, 1975.

The bile acids derived from [4-^{14}C]cholesterol administered intracardially to rats with cannulated bile ducts were identified and quantitated. Over a period of 28 days about 90% of the administered ^{14}C was found in bile of which 73% was retained in the biliary acid fraction. [7β-^{3}H]cholic acid, α-muri[3β-^{3}H]cholic acid, β-muri[3β-^{3}H]cholic acid and litho[3β-^{3}H]cholic acid were prepared with specific activities of about 30 µCi/mg by reduction of appropriate ketonic precursors with $NaB^{3}H_4$ and were added to the biliary acid fraction. After separation and purification of the bile acids, cholic, chenodeoxycholic, α- and β-muricholic acids accounted for 70, 16, 7.5 and 6.1%, resp. of the ^{14}C in the biliary acid fraction. The specific activities of these isolated ^{14}C-labeled acids were almost identical. Lithocholic acid accounted for a maximum of 0.2% and ursodeoxycholic acid and 7-oxolithocholic acid could account for no more than 2% of the biliary ^{14}C. Gas-liquid chromatography on 3% OV-17 of the trimethylsilyl ether derivatives of the methyl esters of the common bile acids of rat bile results in their complete separation and provides a convenient means of estimating the relative proportions of these acids in rat bile. By this method, the relative amounts of the four major acids, cholic, chenodeoxycholic, α- and β-muricholic acids were 63, 20, 8 and 6%, resp.

0931 EFFECT OF DIETARY BILE ACIDS, CHOLESTEROL, AND β-SITOSTEROL UPON FORMATION OF COPROSTANOL AND 7-DEHYDROXYLATION OF BILE ACIDS BY RAT. *(E.)* Cohen, B. I.; Raicht, R. F.; Mosbach, E. H. (Pub. Health Res. Inst. City New York, N.Y.). *Lipids* 9(12):1024-1029, 1974.

Male Sprague-Dawley rats were used to study: 1) the effect of feeding bile acids (sodium taurochenodeoxycholate and sodium taurocholate) and sterol (cholesterol and β-sitosterol) upon coprostanol formation; 2) the transformation of β-sitosterol to coprositostanol; and 3) the ratio of primary to secondary bile acids in feces. Animals were fed the experimental diet for 10 days and 2-day samples of feces collected on days 6, 8 and 10 were analyzed by thin-layer and gas-liquid chromatography. On a stock diet rat chow supplemented with 5% corn oil, the rats excreted 14.5 mg/day of total neutral sterol. Coprostanol comprised 35% (5 mg/day) of this fraction. When the diet was supplemented with 0.5% sodium taurochenodeoxycholate, the amount of coprostanol in the feces remained the same as in the controls (3.2 mg/day, 32%). The addition of 0.5% sodium taurocholate to the diet resulted in a 5-fold reduction of coprostanol formation (0.6 mg/day, 8%). When 1.2% cholesterol was added to the stock diet, the amount of coprostanol present in the feces decreased to an average of 11% compared to controls, but the absolute amount formed was greater (35 mg/day). On a diet enriched with 0.8% β-sitosterol, the rats, on the average, converted 24% of the cholesterol to coprostanol. Feeding diets enriched with sodium taurocholate reduced the 7-dehydroxylation of primary bile acids in the feces by 28% and 42%, resp. The conversion of primary bile acids to secondary bile acids in the feces of control, cholesterol, and β-sitosterol fed rats was the same (64%).

These results show that large amounts of dietary sterols and bile acids have different effects on intestinal bacterial degradation of cholesterol, plant sterols and bile acids which may be of significance since previous studies have implicated the interaction of sterols and bile acids with the intestinal wall as an etiologic agent in colon cancer.

0932 INFLUENCE OF SYNTHETIC CONJUGATES OF CHOLIC ACID ON CHOLESTEREMIA IN RATS. *(E.)* Story, J. A.; Tepper, S. A.; Kritchevsky, D. (Wistar Inst. Anat. Biol., Philadelphia, Pa.). *J. Nutr.* 104(9): 1185-1188, 1974.

The effects on serum and liver cholesterol levels in male Wistar rats of 2 naturally occurring conjugates of cholic acid (taurocholic and glycocholic acids) and 4 synthetic conjugates (glutamocholic, aspartocholic, cysteocholic and cysteinocholic acids) (0.5% of the diet), in combination with cholesterol (0.5% of the diet) were investigated and hydrolysis of these conjugates by cholylglycine hydrolase was measured. Although cholesterol alone did not cause cholesteremia, combination with cholic acid or with any of its conjugates (except aspartocholate) resulted in significantly higher serum-liver cholesterol pools (15-70%) than in controls. The aspartocholic acid-fed group had serum and liver cholesterol levels significantly lower than the cholic acid:cholesterol-fed animals but similar to those of controls. All conjugates of cholic acid were hydrolized to the same extent (77-87%) by cholylglycine hydrolase except for aspartocholic (36%) and cysteinocholic (42%) acids. There thus appears to be a relation between the ability of a cholic acid conjugate to produce elevated serum and/or liver cholesterol levels in rats and the degree to which it is hydrolized by the intestinal microflora.

0933 MECHANISM OF SECRETION OF BILIARY LIPIDS: I. ROLE OF BILE CANALICULAR AND MICROSOMAL MEMBRANES IN THE SYNTHESIS AND TRANSPORT OF BILIARY LECITHIN AND CHOLESTEROL. *(E.)* Gregory, D. H.; Vlahcevic, Z. R.; Schatzki, P.; Swell, L. (VA Hosp., Richmond, Va.). *J. Clin. Invest.* 55(1):105-114, 1975.

The role of bile canalicular and microsomal membranes in the synthesis and transport of biliary lipids was studied in isolated perfused male Wistar rat livers with cannulated common bile ducts. Labeled lecithin precursors ([^{3}H]-palmitic acid, [^{14}C]linoleic acid, [^{3}H]choline, and $^{32}PO_4$) and a cholesterol precursor ([^{3}H]mevalonic acid) were administered with and without sodium taurocholate (10 mg/hr). The incorporation pattern of these labeled precursors into linoleyl and arachidonyl lecithins and cholesterol fractions of microsomes, bile canaliculi, and bile were examined at 30-min intervals up to 90 min. Marker enzymes and electron microscopy indicated that isolated subfractions of plasma membranes were enriched with bile canaliculi (<10% microsomal contamination). Taurocholate significantly stimulated the incorporation of $^{32}PO_4$, [^{3}H]choline, [^{3}H]palmitic acid, and [^{14}C]linoleic acid into linoleyl and arachidonyl lecithin with parallel incorporation curves for microsomal and bile canalicular membranes throughout the 90-min study period. During the 30-60-min period, however, these same lecithin fractions in bile significantly exceeded the specific activity of the membrane lecithins. The enzyme CDP-choline diglyceride transferase was virtually absent from canaliculi relative to microsomes, indicating that canaliculi lack the capacity for *de novo* lecithin synthesis. Incorporation of [^{3}H]mevalonic acid into membranous and biliary cholesterol followed a pattern similar to that for lecithin. These data provide evidence that (1) biliary lecithin and cholesterol are derived from a microsomal subpool regulated by the flux of enterohepatic bile acids, (2) the role of the bile canalicular membranes with respect to biliary lipids is primarily transport rather than synthesis, and (3) lecithin and cholesterol are transported together from microsomes to bile. The findings are consistent with the existence of a cytoplasmic lipid complex within the hepatocyte which is actively involved in the intermembrane transport of biliary lipid.

0934 STIMULATION OF HEPATIC BILE SECRETION BY DOPAMINE. *(E.)* Harty, R. F.; Rose, R. C.; Nahrwold, D. L. (Coll. Med., Pennsylvania State Univ., Hershey). *J. Surg. Res.* 17(5):359-363, 1974.

The effect of dopamine on hepatic bile secretion and the influence of propranolol on the biliary response to dopamine was studied in 4 mongrel dogs which had undergone cholecystectomy, monor pancreatic duct ligation, and placement of Thomas cannulas in the most dependent portion of the stomach and in the duodenum opposite the opening of the common bile duct. Two hr after the start of sodium taurocholate infusion (20 μM/min), one of the following was added and was administered for 2.5 hr: dopamine HCl, 20 μg/kg min; propranolol HCl, 3.3 μg/kg min after an initial loading dose of 250 μg/kg; dopamine plus propranolol in the above dosages; or dopamine (20 μg/kg min) plus propranolol, 6.6 μg/kg min after an initial loading dose of 500 μg/kg. Each of these 4 experiments was performed 3 times on each dog in random order. Bile was collected throughout each experiment and separated into 15-min samples. Dopamine significantly increased bile flow rate from a mean control value of 2.9 ml/15 min to a mean peak value of 4.4 ml/15 min within 60 min after the dopamine infusion was begun. Mean bicarbonate concentration increased from 14.8 mEq/liter to 36.2 mEq/liter, mean bicarbonate output from 45 μEq/15 min to 160 μEq/15 min, chloride concentration from 68.5 mEq/liter to 79.8 mEq/liter, and chloride output from 209 μEq/15 min to 342 μEq/15 min. Dopamine had no effect on bile salt output. Propranolol had no effect on bile flow rate, bicarbonate concentration, bicarbonate output, chloride concentration, or chloride output. When propranolol was administered simultaneously with dopamine it prevented the increase in bile flow observed with dopamine alone but did not prevent the increase in bicarbonate output. It is postulated that dopamine, through its action on adenyl cyclase and cyclic AMP, mediates the release of gastrointestinal hormones or acts as a local gastrointestinal hormone.

0935 BILIARY EXCRETION KINETICS OF PHENOLPHTHALEIN GLUCURONIDE AFTER INTRAVENOUS AND RETROGRADE BILIARY ADMINISTRATION. *(E.)* Gustafson, J. H.; Benet, L. Z. (Sch. Pharm., Univ. California, San Francisco). *J. Pharm. Pharmacol.* 26(12):937-944, 1974.

Biliary reabsorption phenomena were examined for phenolphthalein glucuronide in male Sprague-Dawley rats by measuring biliary and plasma concentrations after i.v. and retrograde biliary administration. Doses of 10-20 mg/kg (20-40 nM/kg) of phenolphthalein glucuronide were administered to 14 rats i.v. and to 12 rats *via* the biliary route. In both cases, 8-10 blood samples (0.4 ml) were withdrawn at selected times up to 20 min after administration of the drug. These were immediately centrifuged and aliquots of plasma withdrawn from the supernatant for assay. Total bile was collected at selected times for up to 3 hr after administration of the drug. A spectrophotometric method, capable of measuring phenolphthalein glucuronide in amounts as small as 4 nM/100 µl of plasma, was developed. There was a significant concentration of phenolphthalein glucuronide in the systemic blood after glucuronide administration by either route and the kinetics of elimination of glucuronide were similar. The plasma availability of biliary infused doses was over 45% of the availability from the i.v. doses based on area under the curve calculations for the average plasma level-time curves. The results demonstrate the need to sample the plasma as well as the bile before any conclusion can be drawn about reabsorption of a compound from the biliary ducts.

0936 EFFECT OF MICROSOMAL ENZYME INDUCERS ON THE BILIARY EXCRETION OF CARDIAC GLYCOSIDES. *(E.)* Klaassen, C. D. (Univ. Kansas Med. Cent., Kansas). *J. Pharmacol. Exp. Ther.* 191(2):201-211, 1974.

The abilities of 4 microsomal enzyme inducers to decrease toxicity and enhance excretion of digitoxin, digoxin or ouabain were compared. Male Sprague-Dawley rats were injected i.p. daily for 4 days with (50 mg/kg) phenobarbital, pregnenolone-16α-carbonitrile (PCN, 75 mg/kg), spironolactone (75 mg/kg), or 3-methylcholanthrene (20 mg/kg) and then given digitoxin (2.5 mg/kg), digoxin (7 mg/kg) or ouabain (5 mg/kg) i.v. The enzyme inducers were made up in propylene glycol to deliver the dosages in a volume of 5 ml/kg. The controls were injected with plain propylene glycol. All microsomal enzyme inducers (especially 3-methylcholanthrene) significantly decreased the time between loss and return of the righting reflex after i.v. injection of zoxazolamine solution (4 ml/kg). PCN decreased the toxicity of digitoxin the greatest as shown by increasing its LD_{50} from 7.4 to 15.8; that of digitoxin and ouabain were increased from 23 to 29 and from 16.1 to 20.1, resp. Spironolactone increased the LD_{50} of digitoxin to 11.5, that of digitoxin to 31 and that of ouabain to 18.5. Phenobarbital increased the LD_{50} of ouabain to 20.1, and had no effect on the other 2 glycosides. 3-Methylcholanthrene lowered the LD_{50} of ouabain slightly and had no effect on the other 2. The plasma disappearance of digitoxin was greatly enhanced by spironolactone and PCN as shown by a 1/3 and 1/6 decrease, resp., in the plasma concentrations. Both also increased biliary excretion of digitoxin 5 and 10 times, resp., that of controls. Similar effects were observed with digitoxin for these 2 enzyme inducers, but slightly lesser effects with ouabain. Phenobarbital increased the plasma disappearance and biliary excretion only of ouabain; 3-methylcholanthrene, in contrast, was near or below control values for all 3 glycosides. These results show there is a marked difference among enzyme inducers in their abilities to decrease toxicity and enhance plasma concentration and biliary excretion of cardiac glycosides. Also, the results support the hypothesis that the enzyme inducers protect by inducing biliary excretion rather than by simply transforming the glycosides to more polar metabolites.

0937 REDUCED RESPONSE TO PHENOBARBITAL BY THE LIVER OF RATS FED A CHOLINE-DEFICIENT DIET. *(E.)* Saito, R.; Estes, L. W.; Lombardi, B. (VA Hosp., Pittsburgh, Pa.). *Biochim. Biophys. Acta* 381(1):185-194, 1975.

The effects of choline deprivation on liver function and lipid content following phenobarbital stimulation were studied in male Sprague-Dawley rats. Groups of 4 rats were fed a normal laboratory chow diet, a semipurified choline-supplemented diet, or a semipurified choline deficient diet for 5 days. Phenobarbital was also injected i.p. daily for 5 days and liver protein, lipid and cytochrome P-450 content as well as aminopyrine and ethylmorphine demethylase activities were determined. Phenobarbital-treated rats maintained on the choline-supplemented diet showed no differences from those fed normal chow with respect to content of cellular constituents and demethylase activities. The normal phenobarbital response was, however, reduced in the livers of rats fed a choline-deficient diet. There was less accumulation of smooth endoplasmic reticulum membranes as determined by electron microscopy as well as decreased levels of cellular triglycerides, cholesterol esters, lecithins, proteins, glycogen and total phospholipids. Cytochrome P-450 and demethylase activities were only 36-58% those of control or choline-supplemented rats. It thus appears that dietary choline plus adequate lecithin synthesis are necessary for induction of hepatic microsomal mixed-function oxidases and the concomitant accumulation of smooth endoplasmic reticulum in liver cells.

0938 COMPARISON OF TWO TYPES OF INTRAHEPATIC JAUNDICE IN RATS WITH BILE DUCT LIGATION. *(E.)* Czygan, P.; Greim, H.; Hutterer, F.; Schaffner, F.; Popper, H. (Mt. Sinai Sch. Med., City Univ. New York, N.Y.). *Acta Hepatogastroenterol. (Stuttg.)* 21(5):339-345, 1974.

The effects of intrahepatic cholestasis induced by norethandrolone, α-naphthylisothiocyanate (ANIT) and by bile duct ligation on hepatic bile acids, microsomal enzymes, microsomal protein and hepatic ultrastructure were compared in male Sprague-Dawley rats.

Compared to a control serum bilirubin of 0.1 mg%, norethandrolone (100 mg/kg i.p. for 3 days) caused an increase to a non-icteric level of 0.7 mg%; serum bilirubin 48 hr after a single ANIT administration (200 mg/kg p.o.) was 9.9 mg% and those 24, 48 and 72 hr after bile duct ligation were 3.4, 8.1 and 8.3 mg%, resp., all icteric levels. Bile duct ligation resulted in disappearance of secondary deoxycholic and hyodeoxycholic acids after the 1st day, a 3-fold increase in chenodeoxycholic acid by the 2nd day, a 2-fold increase in cholic acid by the 3rd day, and 10-20-fold increases in ursodeoxycholic and β-muricholic acids by the 3rd day. Norethanodrolone induced changes which qualitatively resembled those induced by ligation, with 2-fold increases in cholic and chenodeoxycholic acids, a 4-fold increase in β-muricholic acid and no change in deoxycholic acid. By contrast, ANIT produced a 10-fold increase in β-muricholic acid with disappearance of all hydroxybile acids. Cytochrome P-450 content, aminopyrine demethylase and aniline hydroxylase were decreased in all 3 systems. Morphologic changes induced by norethandrolone and bile duct ligation were similar although less severe in the former; centrilobular bile canalicular dilatation, edematous microvilli, Golgi dilatation and curled mitochondrial cristae. By contrast, ANIT produced peripheral bile canicular dilatation, bleb formation on the sinusoidal and canalicular surfaces of hepatocytes and on the luminal surface of bile duct cells, breaks in the outer mitochondrial membranes, and smooth endoplasmic reticulum hypertrophy. It thus appears that there are at least 2 mechanisms by which intrahepatic jaundice may be induced in rats.

0939 THE ARTERIALIZATION OF THE LIVER IN CHOLOSTATIC [SIC!] CIRRHOSIS AND ITS EVOLUTION. *(E.)* Papadimitriou, T. (no affil.). *Clin. Eur.* 12(5):467-475, 1974.

Changes in the hepatic arteries occurring in cholestatic cirrhosis were studied by arteriography and histology of post-sacrifice specimens from dogs. Experimental groups consisted of: 1) 6 dogs with their common bile ducts ligated for 30-75 days, and 2) 9 dogs in which the occlusion was maintained for 45-60 days then released for an additional 60-90 days. Another group of 10 dogs served as controls. After 30 days of occlusion, histologic sections showed: increased periportal connective tissue, occasional leukocytic infiltration, widening and hyperemia of the vessels and inspissated bile within bile capillaries and hepatic cells. Cirrhosis and interlobular connective tissue were present after 45 days. After 75 days of obstruction, cirrhosis, marked hyperplasia of interlobar connective tissue, pseudolobules and hepatic cell degeneration were noted. Arteriograms showed an initial increase in the number and width of hepatic arteries and their tributaries with decreased artery caliber and inability to visualize them past the 3rd subdivision in stages of advanced cirrhosis. Hepatic arteriograms and histologic sections of animals in whom the biliary obstruction had been released for 60-90 days showed similar changes including widening of the arterial network in early cases and narrowing in advanced cases of cirrhosis. These results show that the hepatic changes caused by prolonged cholestasis in dogs do not regress, but actually may progress, following release of obstruction.

0940 THE ENSUING CHANGES OF THE HEPATIC VESSELS AND THE HEPATIC PARENCHYMA FOLLOWING COMPLETE EXTRA-HEPATIC OCCLUSION OF THE BILIARY TREE AND THEIR EVOLUTION AFTER THE RELEASE OF THE CHOLESTASIS (EXPERIMENTAL STUDY). *(E.)* Papadimitriou, T. (no affil.). *Clin. Eur.* 12(5):476-490, 1974.

The effects of cholestasis on the changes in hepatic vessels and parenchyma were studied by angiography and histology in 2 groups: 1) 16 dogs whose common bile ducts were ligated for 30-75 days and 2) 9 dogs obstructed for 45-60 days followed by release of the obstruction for 60-90 days. Another group of 10 dogs served as controls. Arteriography revealed progressive widening and increased tortuosity of the hepatic arterial branches which were visible to the liver border after 75 days. Portal venography showed narrowing, increased tortuosity, premature interruptions and porto-portal communications after 75 days. Hepatic venograms showed similar changes with porto-venous communications. Cholangiograms revealed marked biliary distention with increased tortuosity and number of tributaries. Release of biliary obstruction did not prevent progression to hepatic cirrhosis; all animals developed blood-stained ascitic fluid in their peritoneal cavities. Hepatitic changes and fatty liver degeneration were common. Arteriograms, portal and hepatic venograms and cholangiograms showed changes similar to those seen in animals with occluded bile ducts.

0941 HAEMOLYSIS IN EXPERIMENTAL CHOLESTASIS: POSSIBLE ROLE OF ERYTHROCYTE SIALIC ACID. *(E.)* Powell, L. W.; LaMont, J. T.; Isselbacher, K. J. (Massachusetts Gen. Hosp., Boston). *Gut* 15 (10):794-798, 1974.

The relationship between hemolysis, erythrocyte cell membrane neuraminic (sialic) acid content and hepatic neuraminidase activity was studied in adult male Fischer rats with cholestatic jaundice induced by ligation of the common bile duct. The extent of release of neuraminic acid *in vitro* from normal rat erythrocytes was directly proportional to the amount of *Vibrio cholerae* neuraminidase added (up to 50 U/ml). The enzyme had no significant proteolytic effect on the cell membrane, and no significant qualitative differences between the major cellular protein bands was seen between control and enzyme-treated cells. *In vivo* ^{51}Cr-labeled erythrocyte survival as determined in unligated rats was inversely related to the neuraminidase concentration used to pretreat *in vitro* and also to the amount of neuraminic acid released during this treatment. Splenic sequestration of neuraminidase-pretreated erythrocytes was also significantly enhanced in unligated rats. The degree of hemolysis noted above was comparable to that previously found after 12 days in bile duct ligated rats. Common bile duct ligation in the present experiments resulted in a significant increase in hepatic neuraminidase activity as well as a decrease in erythrocyte neuraminic acid content. These results

are consistent with the hypothesis that one or more neuraminidases present in increased amounts in bile duct-ligated livers cause removal of neuraminic acid from circulating erythrocytes resulting in splenic sequestration of these cells.

0942 ROLE OF THE MALATE-ASPARTATE SHUTTLE IN THE METABOLISM OF ETHANOL IN VIVO. *(E.)* Nordmann, R.; Petit, M. A.; Nordmann, J. (INSERM, Paris, France). *Biochem. Pharmacol.* 24(1):139-143, 1974.

The role of the malate-aspartate shuttle pathway in ethanol oxidation was studied in female Sprague-Dawley rats. Animals received 4 i.p. injections of aminooxyacetate (0.275 mM/kg) at 45 min intervals with a single i.p. injection of ethanol (10 mM/kg) 10 min after the first aminooxyacetate dose. Liver specimens taken under anesthesia 30 min after ethanol injection were analyzed for metabolites; the rate of disappearance of ethanol from the blood was also determined. Compared to saline-injected controls, the rate of ethanol elimination from blood was decreased by 50% in aminooxyacetate-treated animals. Under these conditions, liver aspartate aminotransferase activity was reduced by 90% during the first hr, decreasing to 60% during the following 2 hr. Aminooxyacetate alone produced a 5-fold increase in the hepatic lactate:pyruvate ratio and a 58% decrease in the β-hydroxybutyrate:acetoacetate ratio. Ethanol alone resulted in a 3-fold increase in the lactate:pyruvate ratio but had no effect on the β-hydroxybutyrate:acetoacetate ratio. Combined administration of aminooxyacetate and ethanol enhanced the aminooxyacetate-induced reduced state of the extramitochondrial space (lacate:pyruvate = 10) but did not significantly alter the intramitochondrial oxidized state induced by aminooxyacetate alone. Hepatic α-glycerophosphate levels were enhanced by both aminooxyacetate alone and ethanol alone whereas aminooxyacetate produced a 70% decrease and ethanol a 35% increase in hepatic aspartate. These data show that the malate-aspartate shuttle is inhibited by aminooxyacetate, thus promoting ethanol-induced disturbances of the cytosol redox state; further, the data provide evidence for the *in vivo* involvement of the malate-aspartate shuttle as a rate limiting factor in the transport of reducing equivalents resulting from ethanol oxidation in liver.

0943 ETHANOL-INDUCED CHANGES IN METHIONINE METABOLISM IN RAT LIVER. *(E.)* Finkelstein, J. D.; Cello, J. P.; Kyle, W. E. (VA Hosp., Washington, D.C.). *Biochem. Biophys. Res. Commun.* 61(2):525-531, 1974.

The effect of ethanol (4.4 gm/kg/day p.o.) on methionine metabolism in the livers of male Sprague-Dawley rats fed a protein-restricted diet was studied. Ethanol administration for 3-10 days had little effect on the activities of liver s-adenosylmethionine synthetase, s-adenosylhomocysteine synthetase, 5-methyltetrahydrofolate-homocysteine methyltransferase, betaine-homocysteine methyltransferase, cystathione synthetase and cystathionase of animals fed a diet containing 26% or 55% casein (except for a 50% increase in s-adenosylmethionine synthetase in the 26% casein group). However, 5-methyltetrahydrofolate-homocysteine methyltransferase activity was decreased by 20-40%, s-adenosylmethionine synthetase, betaine methyltransferase, and cystathione synthetase activities were increased by 40-170%, and s-adenosylhomocysteine synthetase and cystathionase acitvities were unchanged in livers of rats fed a low (3.5%) casein diet. Alcohol-treated rats gained weight at the same rate as controls fed a similar diet with isocaloric substitution of sucrose for ethanol. Enzyme activities in brain or kidney were unaffected by ethanol. These changes represent a reversal of the normal adaptive response to protein restriction. The resulting impairment in methionine metabolism could explain the ethanol-induced increase in the dietary lipotrope requirement previously observed.

0944 LONG TERM CELL CULTURE OF RAT LIVER EPITHELIAL CELLS RETAINING SOME HEPATIC FUNCTIONS. *(E.)* Chessebeuf, M.; Olsson, A.; Bournot, P.; Desgres, J.; Guiguet, M.; Maume, G.; Maume, B. F.; Perissel, B.; Padieu, P. (Group Biochem. Cell. Differ., Univ. Dijon, France). *Biochimie* 56(10):1365-1379, 1974.

0945 VIABILITY OF THE ISOLATED LIVER AFTER ITS CLINICAL UTILIZATION: EXPERIMENTAL STUDY OF VARIOUS PRESERVATION TECHNIQUES. *(Sp.)* de Miguel, E.; Goiti, J. J.; Fresneda, V.; Castillo-Olivares Gosalvez, J. L. (Puerta de Hierro Clin., Madrid, Spain). *Rev. Esp. Enferm. Apar. Dig.* 44(4):325-342, 1974.

0946 TESTING THE VITALITY OF ISOLATED RAT LIVER CELLS. *(Ger.)* Hopf, U.; Meyer zum Buschenfelde, K. H.; Groth, U.; Freudenberg, J. (2nd Med. Clin., Univ. Mainz, Germany). *Res. Exp. Med. (Berl.)* 163(3):199-209, 1974.

0947 EVALUATION OF METHODS FOR SPECIFIC LOADING OF KUPFFER CELL LYSOSOMES WITH HEAVY COLLOIDAL PARTICLES. *(E.)* Arborgh, B.; Berg, T.; Ericsson, J. L. E. (Sabbatsberg's Hosp., Karolinska Inst., Stockholm, Sweden). *Acta Pathol. Microbiol. Scand.* [B] 82(6):747-759, 1974.

0948 EXPERIMENTAL ORTHOTOPIC LIVER TRANSPLANTATION: ENHANCED SURVIVAL BY REDUCTION OF NONIMMUNOLOGIC COMPLICATIONS. *(E.)* Schalm, S. W.; Terpstra, J. L.; Popescu, D. T.; Krom, R. A. F.; Achterberg, J. (Univ. Leyden, Holland). *Gastroenterology* 67(4):826, 1974.

0949 ORTHOTOPIC LIVER TRANSPLANTATION AFTER PRESERVATION BY PROLONGED HYPOTHERMIA. *(Por.)* da Cunha, J. E. M.; Machado, M. C. C.; Bacchella, T.; Bevilacqua, R. G.; Margarido, N. F.; Goncalez, Y.; Abdo, E. E.; Goncalvez, E. L. (Fac. Med., Univ. Sao Paulo, Brazil). *Rev. Hosp. Clin. Fac. Med. Sao Paulo* 29(2):90-97, 1974.

0950 COMPARATIVE STUDIES OF IMPEDANCE MEASUREMENT IN THE ORTHOTOPICALLY TRANSPLANTED LIVER. *(Ger.)* Rigler, B.; Lanner, G.; Hermann, W.; Stenzl, W. (Neurosurg. Clin., Univ. Graz, Austria). *Langenbecks Arch. Chir.* 336(2):173-179, 1974.

0951 IMPEDANCE MEASUREMENT: COMPARATIVE INVESTIGATIONS DURING ORTHOTOPIC LIVER TRANSPLANTATION. *(E.)* Rigler, B.; Lanner, G.; Herrmann, W.; Stenzl, W. (Clin. Surg. Neurosurg., Univ. Graz, Austria). *Eur. Surg. Res.* 6(1):76, 1974.

0952 ORTHOTOPIC LIVER TRANSPLANTATION AFTER PRESERVATION WITH COLLINS OR SACKS' SOLUTION. *(E.)* Otte, J. B.; Saliez, A.; Polspoel, D.; Lambotte, L. (Lab. Exp. Surg., Louvain, Belgium). *Eur. Surg. Res.* 6(1):86-87, 1974.

0953 HEMODYNAMICS OF THE GRAFT AS FACTOR OF SUCCESS IN LIFE SUPPORTING HETEROTOPIC LIVER TRANSPLANTATION. *(E.)* Lecompte, Y.; Grange, D.; de Riberolles, C.; Brunet, A. M.; Bismuth, H. (INSERM, Paul Brousse Hosp., Villejuif, France). *Eur. Surg. Res.* 6(1):75-76, 1974.

0954 CANINE INTRATHORACIC HEPATIC HOMOGRAFT. A LIFE-SUPPORTING PROCEDURE. *(E.)* LeCompte, Y.; de Riverolles, C.; Grange, D.; Bfunet, A. M.; Bismuth, H. (INSERM, Paul Brousse Hosp., Villejuif, France). *Arch. Surg.* 109(6):809-811, 1974.

0955 LONG-TERM PRESERVATION OF THE CANINE LIVER USING AUXILIARY AUTOTRANSPLANTATION AS A FUNCTIONAL TEST. *(E.)* Slapak, M.; Maki, T. (Dep. Surg., Univ. Cambridge, England). *Eur. Surg. Res.* 6(1):87, 1974.

0956 LIVER CELL MEMBRANE POTENTIALS (CMP) AND MEMBRANEPOTENTIOGRAMS (MPG); THEIR USE IN LIVER PRESERVATION STUDIES AND LIVER TRANSPLANTATION. *(E.)* Paloheimo, M.; Karrinaho, T.; Lempinen, M. (Univ. Cent. Hosp., Helsinki, Finland). *Eur. Surg. Res.* 6(1):88, 1974.

0957 PROLONGATION OF LIVER ALLOGRAFT SURVIVAL BY DONOR-SPECIFIC SOLUBLE TRANSPLANTATION ANTIGENS AND ANTIGEN-ANTIBODY COMPLEXES. *(E.)* Myburgh, J. A.; Smit, J. A. (Univ. Witwatersrand Med. Sch., Johannesburg, South Africa). *Transplantation* 19(1):64-70, 1975.

0958 HYPOXIC DAMAGE OF THE LIVER DUE TO ITS METABOLIC CHANGES DURING ABDOMINAL OPERATIONS IN RATS AND ITS PROBABLE MECHANISM. *(E.)* Gaal, J. G.; Meszaros, M.; Nagy, L.; Guba, F.; Sandor, P. (Budapest Univ. Med. Sch., Hungary). *Eur. Surg. Res.* 6(Suppl. 1):51-52, 1974.

0959 THE EFFECT OF HYPOXIA ON THE FUNCTION OF THE ISOLATED PERFUSED PIG LIVER. *(E.)* Rabol, A.; Hansen, F. V.; Keiding, S.; Tygstrup, N.; Tonnesen, K.; Winkler, K. (Kommunehosp., Copenhagen, Denmark). *Digestion* 10(4/5):375-376, 1974.

0960 SENSITIVITY OF THE VASCULAR ENDOTHELIUM OF LIVER SINUSOIDS TO ISCHEMIA AND PRESERVATION PROCEDURES. *(E.)* Olszewski, W.; Borowicz, J.; Olszewska, K. (Polish Acad. Sci., Warsaw). *Eur. Surg. Res.* 6(1):88-89, 1974.

0961 RETICULOENDOTHELIAL FUNCTION AND ENDOTOXEMIA FOLLOWING HEPATIC ISCHEMIA IN THE PRIMATE. *(E.)* Holper, K.; Olcay, I.; Kitahama, A.; Trejo, R. A.; Miller, R.; Brettschneider, L.; Diluzio, N. R.; Drapanas, T. (German Heart Cent., Munich). *Eur. Surg. Res.* 6(Suppl. 1):53, 1974.

0962 EFFECTS OF AMINO ACIDS ON PROTEIN SYNTHESIS BY CELLULAR AND SUBCELLULAR PREPARATIONS FROM ISCHAEMIC LIVERS. *(E.)* Cajone, F.; Schiaffonati, L. (Cent. Cell. Pathol., Univ. Milan, Italy). *Br. J. Exp. Pathol.* 55(6):575-582, 1974.

0963 LONG-TERM MORPHOLOGICAL, HISTOCHEMICAL AND HISTOENZYMOLOGICAL ASPECTS OF RAT LIVER FOLLOWING SUBTOTAL HEPATECTOMY OR REPEATED SUBTOTAL HEPATECTOMY. *(Rum.)* Preda, V.; Craciun, O. (Inst. Med. Pharm., Cluj, Rumania). *Cluj. Med.* 46(3):559-571, 1973.

0964 AUTOSYNCHRONIZATION OF RAT LIVER CELLS WITH ENDOGENOUS CORTICOSTERONE AFTER PARTIAL HEPATECTOMY. *(E.)* Desser-Wiest, L. (Cancer Res. Inst., Univ. Vienna, Austria). *Cell Tissue Kinet.* 8(1):1-9, 1975.

0965 AN ELECTRON MICROSCOPIC STUDY OF THE EFFECTS OF PORTACAVAL SHUNTS ON THE ULTRASTRUCTURE OF THE RAT LIVER AFTER PARTIAL HEPATECTOMY. *(E.)* Marks, C.; Markey, C.; Dyer, R.; Vaupel, M. R. (Louisiana State Univ. Sch. Med., New Orleans). *Am. J. Surg.* 129(2):156-162, 1975.

0966 PORTACAVAL SHUNT IN THE PIG. 2. EVALUATION OF FUNCTIONAL CHANGES OF THE LIVER BY A COMPARTMENTAL BROMSULPHTHALEIN (BSP) TEST. *(E.)* Baker, P. R.; Cuschieri, A.; Hanson, C. (Dep. Surg., Univ. Liverpool, England). *J. Surg. Res.* 17(6):397-403, 1974.

0967 INFLUENCE OF PORTACAVAL ANASTOMOSIS ON CLOTTING FACTORS IN RATS. *(E.)* Brunswig, D.; Grun, M.; Liehr, H. (Dep. Med., Univ. Wurzburg, Germany). *Digestion* 10(4/5):339, 1974.

0968 EFFECT OF THAM, ISOPRENALINE AND PROPANOLOL ON HEPATIC BLOOD FLOW AND RESISTANCES AFTER IN, AND OUTFLOW OCCLUSION OF THE LIVER. RELATIONS WITH SPLANCHNIC SHOCK. *(E.)* Stoitchcov, E.; Kawai, T.; Boissel, P.; Benichoux, R. (no affil.). *Eur. Surg. Res.* 6(Suppl. 1):52-53, 1974.

0969 INFLUENCE EXERTED ON THE LIVER BY INTRAPORTAL INTRODUCTION OF OXYGEN. *(Rus.)* Maliugin, E. F.; Togobetskii, A. S. (N. I. Pirogov 2nd Moscow Med. Inst., USSR). *Farmakol. Toksikol.* 37:183-186, 1974.

0970 BLOOD CHANGES DEVELOPING DURING PERFUSION THROUGH ISOLATED PIG LIVER. *(Pol.)* Jesipowicz, M.; Karski, J.; Kurylcio, L.; Pozarowski, A.; Szczepinska, J.; Dzida, A. (Acad. Med., Lublin, Poland). *Pol. Tyg. Lek.* 29(35):1505-1507, 1974.

0971 BLOOD FLOW REGULATION IN THE LIVER DURING ACUTE PRESSURE INCREASE IN THE PORTAL VEIN OR IN THE VENA CAVA. *(Sl.)* Lutz, J. (Inst. Physiol., Univ. Wurzburg, Germany). *Bratisl. Lek. Listy* 61(3):314-318, 1974.

0972 CONTROLLED ARTERIALIZATION OF THE LIVER. I. EXPERIMENTAL OBSERVATIONS. *(Ger.)* Funovies, J.; Gangl, A.; Horak, W.; Grabner, G.; Kohn, P.; Zaunbauer, F.; Fritsch, A. (1st Surg. Clin., Univ. Vienna, Austria). *Langenbecks Arch. Chir.* 335(4):339-359, 1974.

0973 FETAL HEMOPOIESIS. II. ELECTRON MICROSCOPIC STUDIES ON HUMAN HEPATIC HEMOPOIESIS. *(E.)* Fukuda, T. (Dep. Pathol., Tohoku Univ., Sendai, Japan). *Virchows Arch.* [*Zellpathol.*] 16(3):249-270, 1974.

0974 EFFECT OF HEMODILUTION, HEMOCONCENTRATION AND β-ADRENERGIC STIMULATION ON THE BLOOD FLOW AND MEMBRANE POTENTIAL VALUE OF THE LIVER. *(E.)* Lambotte, L. (Lab. Exp. Surg., Univ. Louvain, Belgium). *Eur. Surg. Res.* 6(3):160-167, 1974.

0975 DEOXYRIBONUCLEIC ACID SYNTHESIS IN REGENERATING RAT LIVER STUDIED BY CYTOFLUOROMETRY IN COMBINATION WITH TRITIATED THYMIDINE AUTORADIOGRAPHY. *(E.)* Fujita, S.; Ashihara, T.; Fukuda, M.; Takeoka, O. (Kyoto Prefect. Univ. Med., Japan). *Acta Histochem. Cytochem.* 7(2):105-111, 1974.

0976 EFFECTS OF ETHANOL AND ETHIONINE ON DNA SYNTHESIS DURING EXPERIMENTAL LIVER REGENERATION. *(E.)* Craig, J. (Univ. South. California Sch. Med., Los Angeles). *J. Stud. Alcohol* 36(1):148-157, 1975.

0977 ROLE OF THE STABILITY OF RIBONUCLEIC ACID TEMPLATE DURING INDUCED LIVER GROWTH. *(E.)* Nievel, J. G. (King's Coll. Hosp. Med. Sch., London, England). *Biochem. Soc. Trans.* 2(6):1255-1256, 1974.

0978 EFFECT OF DEPLETION OF VITAMIN A, FOLLOWED BY SUPPLEMENTATION WITH RETINYL ACETATE OR RETINOIC ACID, ON REGENERATION OF RAT LIVER. *(E.)* Jayaram, M.; Sarada, K.; Ganguly, J. (Indian Inst. Sci., Bangalore). *Biochem. J.* 146(2):501-504, 1975.

0979 LYSOSOME-VACUOLAR SYSTEM REACTIVITY DURING EARLY CELL REGENERATION. *(E.)* Verity, M. A.; Travis, G.; Cheung, M. (Univ. California Cent. Health Sci., Los Angeles). *Exp. Mol. Pathol.* 22(1):73-84, 1975.

0980 POSSIBLE INTERACTIONS BETWEEN THE UREA CYCLE AND SYNTHESIS OF PYRIMIDINES AND POLYAMINES IN REGENERATING LIVER. *(E.)* Fausto, N.; Brandt, J. T.; Kesner, L. (Div. Biol. Med. Sci., Brown Univ., Providence, R.I.). *Cancer Res.* 35(2):397-404, 1975.

0981 FEATURES OF CELL PROLIFERATION IN REGENERATING RAT LIVER. *(Rus.)* Beliaeva, I. D. (Inst. Biol. Med. Chem., Moscow, USSR). *Tsitologiia* 15(10):1297-1300, 1973.

0982 ORGAN CULTURE OF FETAL LIVER. *(E.)* de Belle, R. C.; Blacklow, N. R.; Little, J. M.; Whalen, R.; Lester, R. (Dep. Med., Univ. Pittsburgh, Pa.). *Gastroenterology* 67(4):788, 1974.

0983 RNA AND PROTEIN SYNTHESIS AND THE SIZE OF LIVER CELLS IN THE RAT. *(Rus.)* Zhinkin, L. N.; Nilova, V. K.; Diment, A. V. (Inst. Cytol., Leningrad, USSR). *Tsitologiia* 15(5):543-550, 1973.

0984 CYCLIC NUCLEOTIDES INFLUENCE THE DNA SYNTHESIS OF RAT LIVER CELLS IN PRIMARY CULTURES. *(E.)* Armato, U.; Andreis, P. G.; Draghi, E. (Univ. Padua Med. Sch., Italy). *Digestion* 10(4/5):333-334, 1974.

0985 INCORPORATION RATE OF LEUCINE INTO PROTEINS IN HUMAN LIVER SLICES. *(E.)* Stakeberg, H.; Gustafson, A.; Schersten, T. (Sahlgrenska Hosp., Univ. Goteborg, Sweden). *Eur. J. Clin. Invest.* 4(6):393-398, 1974.

0986 PLASMA ENZYME ACTIVITIES IN RATS WITH DIET-INDUCED ALTERATIONS IN LIVER ENZYME ACTIVITIES. *(E.)* Bolter, C. P.; Critz, J. B. (Dep. Physiol., Univ. West. Ontario, London, Canada). *Experientia* 30(11):1241-1243, 1974.

0987 CIRCADIAN CHANGES IN THE ACTIVITY OF ORNITHINE DECARBOXYLASE IN LIVERS OF RATS INGESTING FOOD IN A CONTROLLED MANNER. *(It.)* Tadolini, B.; Monti, M. G.; Moruzzi, M. S. (Inst. Biochem., Univ. Bologna, Italy). *Boll. Soc. Ital. Biol. Sper.* 49(23):1379-1382, 1973.

0988 NITROREDUCTION OF 5-NITROFURAN DERIVATIVES BY RAT LIVER XANTHINE OXIDASE AND REDUCED NICOTINAMIDE ADENINE DINUCLEOTIDE PHOSPHATE-CYTOCHROME c REDUCTASE. *(E.)* Wang, C. Y.; Behrens, B. C.; Ichikawa, M.; Bryan, G. T. (Univ. Wisconsin Med. Sch., Madison). *Biochem. Pharmacol.* 23(24):3395-3404, 1974.

0989 ADENOSINE DEAMINASE ACTIVITY IN LIVERS OF CONGENITALLY ATHYMIC NUDE MICE. *(E.)* Deutsch, S. I.; Romano, T. J.; Ghosh, N. K.; Cox, R. P. (New York Univ. Sch. Med., N.Y.). *Experientia* 11(30):1247-1248, 1974.

0990 RESOLUTION OF GLUTATHIONE-LINKED ENZYMES IN RAT LIVER AND EVALUATION OF THEIR CONTRIBUTION TO DISULFIDE REDUCTION VIA THIOL-DISULFIDE INTERCHANGE. *(E.)* Eriksson, S.; Askelof, P.; Axelsson, K.; Carlberg, I.; Guthenberg, C.; Mannervik, B. (Arrhenius Lab., Univ. Stockholm, Sweden). *Acta Chem. Scand.* [*B*] 28(8):922-930, 1974.

0991 EVIDENCE FOR A GLUTATHIONE-DEPENDENT INTERCONVERSION OF TWO FORMS OF A THIOLTRANSFERASE FROM RAT LIVER CATALYZING THIOL-DISULFIDE INTERCHANGE. *(E.)* Eriksson, S.; Askelof, P.; Axelsson, K.; Mannervik, B. (Arrhenius Lab., Univ. Stockholm, Sweden). *Acta Chem. Scand.* [*B*] 28(8):931-936, 1974.

0992 DECREASED SYNTHESIS OF 'SECRETED' PROTEINS IN CHICK EMBRYO LIVER CULTURES TREATED BY ESTRADIOL-17-β. *(E.)* Carinci, P.; Bodo, M. A.; Locci, P.; Evangelisti, R. (Inst. Histol. Gen. Embryol., Univ. Perugia, Italy). *Experientia* 30(11): 1243-1244, 1974.

0993 SECRETION OF ALBUMIN AND ANGIOTENSINOGEN (RENIN SUBSTRATE) BY ENZYMATICALLY ISOLATED RAT LIVER CELLS. *(E.)* Weigand, K.; Wernze, H. (Med. Clin., Univ. Wurzburg, Germany). *Digestion* 10(4/5): 385-386, 1974.

0994 MURINE CYTOMEGALOVIRUS-INFECTED LIVER: DEPLETION OF ACID PHOSPHATASE WITH CONCOMITANT INCREASE OF ALKALINE PHOSPHATASE. *(E.)* Mohamed, A. H. (Sch. Dent., Univ. California, Los Angeles). *Microbios* 10(38/39):103-113, 1974.

0995 INVOLVEMENT OF THE LIVER IN THE REGULATION OF TRYPTOPHAN AVAILABILITY. POSSIBLE ROLE IN THE RESPONSES OF LIVER AND BRAIN TO STARVATION. *(E.)* Bloxam, D. L.; Warren, W. H.; White, P. J. (Dep. Pathol., Univ. Toronto, Canada). *Life Sci.* 15(8):1443-1455, 1974.

0996 EFFECT OF ZINC DEFICIENCY ON URINARY EXCRETION OF NITROGENOUS COMPOUNDS AND LIVER AMINO ACID-CATABOLIZING ENZYMES IN RATS. *(E.)* Hsu, J. M.; Anthony, W. L. (VA Hosp., Baltimore, Md.). *J. Nutr.* 105(1):26-31, 1975.

0997 IN VITRO STIMULATION OF APOFERRITIN SYNTHESIS BY IRON. *(E.)* Drysdale, J. W.; Shafritz, D. A. (Tufts Univ. Sch. Med., Boston, Mass.). *Biochim. Biophys. Acta* 383(1):97-105, 1975.

0998 EFFECT OF LOW CALCIUM CONCENTRATION ON THE OXIDATION OF NAD-LINKED SUBSTRATES IN RAT LIVER AND TUMOR MITOCHONDRIA. *(E.)* Eboli, M. L.; Cittadini, A.; Terranova, T. (Inst. Gen. Pathol., Cathol. Univ., Rome, Italy). *Experientia* 30(10):1125-1127, 1974.

0999 SYNTHESIS AND TURNOVER OF LIVER PROTEINS DURING EXTRAHEPATIC CHOLESTASIS IN THE RAT. *(E.)* Lundborg, H.; Hamberger, A. (Sahlgrenska Hosp., Univ. Goteborg, Sweden). *Eur. Surg. Res.* 6(4):219-232, 1974.

1000 THE EFFECT OF SOME INDUCERS OF HEPATIC XENOBIOTIC-METABOLIZING ENZYMES ON THE URINARY EXCRETION OF D-GLUCURONIC ACID METABOLITES IN THE RAT. *(E.)* Lake, B. G.; Longland, R. C.; Hodgson, R. A.; Severn, B. J.; Gangolli, S. D.; Lloyd, A. G. (Br. Ind. Biol. Res. Assoc., Carshalton, England). *Biochem. Soc. Trans.* 3(1):188-191, 1975.

1001 HEPATIC CLEARANCE OF ADENOSINE 3':5'-CYCLIC MONOPHOSPHATE FROM PLASMA IN THE RAT. *(E.)* Strange, R. C.; Percy-Robb, I. W. (R. Infirm., Edinburgh, Scotland). *Biochem. J.* 146(2):509-512, 1975.

1002 CONTROL OF PHOSPHORYLATIVE ACTIVITY IN HUMAN LIVER MITOCHONDRIA THROUGH CHANGES IN RESPIRATORY ENZYME CONTENTS. *(E.)* Ozawa, K.; Honjo, I. (Fac. Med., Kyoto Univ., Japan). *Clin. Sci. Mol. Med.* 48(2):75-82, 1975.

1003 CHARACTERISTICS OF THE INDUCTION OF ALDEHYDE DEHYDROGENASE IN RAT LIVER. *(E.)* Deitrich, R. A.; Troxell, P. A.; Erwin, V. G. (Univ. Colorado Sch. Med., Boulder). *Arch. Biochem. Biophys.* 166(2):543-548, 1975.

1004 INCREASED SYNTHESIS OF LOW-MOLECULAR-WEIGHT NUCLEAR RIBONUCLEIC ACIDS OF RAT LIVER AFTER GAMMA IRRADIATION AND HEPATECTOMY. *(E.)* Fonagy, A.; Hidvegi, E. J. (Frederic Joliot-Curie Natl. Res. Inst. Radiobiol. Radiohyg., Budapest, Hungary). *Biochem. J.* 146(2):357-360, 1975.

1005 AMINE COMPETITION FOR OXIDATION BY RAT LIVER MITOCHONDRIAL MONOAMINE OXIDASE. *(E.)* Houslay, M. D.; Tipton, K. F. (Dep. Biochem., Univ. Cambridge, England). *Biochem. Pharmacol.* 24(5):627-631, 1975.

1006 THE EFFECTS OF L-LEUCINE ON THE SYNTHESIS OF UREA, GLUTAMATE AND GLUTAMINE BY ISOLATED RAT LIVER CELLS. *(E.)* Mendes-Mourao, J.; McGivan, J. D.; Chappell, J. B. (Med. Sch., Univ. Bristol, England). *Biochem. J.* 146(2):457-464, 1975.

1007 DIETARY SUCROSE AND DNA FORMATION IN RAT LIVER AND KIDNEY WITH STARCH. *(E.)* Mohammadiha, H. (Fac. Med., Univ. Tehran, Iran). *Am. J. Clin. Nutr.* 27(11):1204-1211, 1974.

1008 THE EFFECT OF ACUTE URAEMIA ON GLUCONEOGENESIS IN ISOLATED PERFUSED RAT LIVERS. *(E.)* Frohlich, J.; Scholmerich, J.; Hoppe-Seyler, G.; Maier, K. P.; Talke, H.; Schollmeyer, P.; Gerok, W. (Dep. Intern. Med., Univ. Freiburg, Germany). *Eur. J. Clin. Invest.* 4(6):453-458, 1974.

1009 HEPATIC CHOLESTEROL SYNTHESIS FROM MEVALONATE AND SQUALENE IN RATS: EFFECT OF FEEDING CHOLESTEROL SUPPLEMENTED DIET DURING WEANING AND FOLLOWING STARVATION. *(E.)* Johnson, R. C.; Shah, S. N. (Sonoma State Hosp., Eldridge, Calif.). *Lipids* 9(12):962-970, 1974.

1010 THE EFFECTS OF PROPRANOLOL AND PHENOXYBENZAMINE ON THE DECREASE OF GLYCOGEN CONTENT OF RAT LIVER DUE TO SURGICAL OPERATION. *(E.)* Gaal, J. G.; Pogatsa, G.; Nagy, L. (Semmelweis Univ. Med. Sch., Budapest, Hungary). *Eur. Surg. Res.* 6(5):289-294, 1974.

1011 REGULATION OF HEPATIC L-SERINE DEHYDRATASE AND L-SERINE-PYRUVATE AMINOTRANSFERASE IN THE DEVELOPING NEONATAL RAT. *(E.)* Snell, K.; Walker, D. G. (Dep. Biochem., Univ. Birmingham, England). *Biochem. J.* 144(3):519-531, 1974.

1012 AGE-RELATED CHANGES IN GLUCONEOGENESIS AND THE METABOLISM OF ALANINE IN THE PERFUSED LIVER OF NEONATAL RATS. *(E.)* Snell, K.; Walker, D. G. (Dep. Biochem. Univ. Birmingham, England). *Biochem. Soc. Trans.* 3(1):128-129, 1975.

1013 THE CELLULAR DISTRIBUTION OF LIPOSOMES IN THE LIVER OF NEWBORN RATS. *(E.)* de Barsy, T.; Devos, P.; Van Hoof, F. (Lab. Chem. Physiol., Univ. Louvain, Brussels, Belgium). *Biochem. Soc. Trans.* 3(1):159-160, 1975.

1014 CONTROL OF HEPATIC GLYCOGEN METABOLISM IN THE RHESUS MONKEY: EFFECT OF GLUCOSE, INSULIN, AND GLUCAGON ADMINISTRATION. *(E.)* Curnow, R. T.; Rayfield, E. J.; George, D. T.; Zenser, T. V.; De Rubertis, F. (US Army Med. Res. Inst. Infect. Dis., Frederick, Md.). *Am. J. Physiol.* 228(1):80-87, 1975.

1015 ELECTRON MICROSCOPIC OBSERVATIONS ON THE MOUSE LIVER AFTER INTRAVENOUS ADMINISTRATION OF FAT EMULSION. *(E.)* Hatae, T. (Fac. Med., Kyushu Univ., Fukuoka, Japan). *Arch. Histol. Jpn.* 37(2):149-163, 1974.

1016 SUBCELLULAR DISTRIBUTION OF FATTY ACIDS SYNTHESIZED BY RAT LIVER SLICES. *(It.)* Landriscina, C.; De Lucia, I. (Inst. Biochem., Univ. Bari, Italy). *Boll. Soc. Ital. Biol. Sper.* 49(17):1011-1017, 1973.

1017 INHIBITION OF DE NOVO LIPOGENESIS IN THE RAT AFTER INGESTION OF A FOOD ANTIOXIDANT: DITERT-BUTYLHYDROXYTOLUENE. *(Fr.)* Pascal, G.; Terroine, T. (Natl. Inst. Agric. Res., Jouy-en-Josas, France). *C. R. Acad. Sci. [D] (Paris)* 279(5):429-432, 1974.

1018 THE INCORPORATION OF ESSENTIAL PHOSPHOLIPIDS INTO THE ORGANS OF INTACT AND GALACTOSAMINE-INTOXICATED RATS. *(Ger.)* Lekim, D.; Betzing, H. (A. Nattermann Inc., Cologne, Germany). *Arzneim. Forsch.* 24(8):1217-1221, 1974.

1019 HEPATOPANCREATIC AND CARDIOVASCULAR LESIONS IN RATS GIVEN VARIOUS HIGH FAT DIETS. *(Fr.)* Rauber, G.; Cherrier, P.; Debry, G. (Univ. Hosp. Cent., Nancy, France). *C. R. Soc. Biol. [D] (Paris)* 168(2/3):257-253, 1974.

1020 MASS-SPECTROMETRIC INVESTIGATION OF THE EFFECT OF HYDROCORTISONE ON LIPID METABOLISM IN RAT LIVER. *(Rus.)* Sergeev, P. V.; Manusadzhian, V. G.; Seifulla, R. D.; Multanovskii, M. M.; Saksonov, N. P. (N. I. Pirogov 2nd Moscow Med. Inst., USSR). *Farmakol. Toksikol.* 37:455-458, 1974.

1021 EFFECT OF DIETARY FAT AND FATTY ACIDS ON LIVER LIPID ACCUMULATION IN FASTED-REFED RATS. *(E.)* Aoyama, Y.; Yoshida, A.; Ashida, K. (Lab. Nutr. Biochem., Nagoya Univ., Japan). *Agric. Biol. Chem.* 38(10):1791-1796, 1974.

1022 EFFECTS OF DIET ON THE ACTIVITIES OF ENZYMES RELATED TO LIPOGENESIS IN RAT LIVER AND ADIPOSE TISSUE. *(E.)* Roggeveen, A. E.; Geisler, R. W.; Peavy, D. E.; Hansen, R. J. (Sch. Vet. Med., Univ. California, Davis). *Exp. Biol. Med.* 147(2):467-470, 1974.

1023 IMMUNOLOGICAL STUDIES OF β GALACTOSIDASE IN NORMAL HUMAN LIVER AND IN G_{M_1} GANGLIOSIDOSIS. *(E.)* Meisler, M.; Rattazzi, M. C. (Dep. Biochem., State Univ. New York, Buffalo). *Am. J. Hum. Genet.* 26(6):683-691, 1974.

1024 EFFECTS OF FASTING ON THE CONTROL OF FATTY-ACID SYNTHESIS IN HEPATOMA 7777 AND HOST LIVER. ROLE OF LONG-CHAIN FATTY ACYL-CoA, THE MITOCHONDRIAL CITRATE TRANSPORTER AND PYRUVATE DEHYDROGENASE ACTIVITY. *(E.)* Halperin, M. L.; Taylor, W. M.; Cheema-Dhadli, S.; Morris, H. P.; Fritz, I. B. (Dep. Med., Univ. Toronto, Canada). *Eur. J. Biochem.* 50(3):517-522, 1975.

1025 EFFECT OF DIETARY COPPER-DEFICIENCY ON THE DESATURATION OF STEARIC ACID BY RAT LIVER MICROSOMAL FRACTIONS. *(E.)* Wahle, K. W. J.; Davies, N. T. (Rowett Res. Inst., Aberdeen, Scotland). *Biochem. Soc. Trans.* 2(6):1283-1285, 1974.

1026 HORMONAL REGULATION OF THE ACTIVITY OF THE FATTY ACID SYNTHESIZING SYSTEM AND OF THE MALIC ENZYME CONCENTRATION IN LIVER CELLS. *(E.)* Goodridge, A. G. (Banting Best Dep. Med. Res., Toronto, Canada). *Fed. Proc.* 34(2):117-123, 1975.

1027 CHANGES IN HEPATIC FATTY ACID DEGRADATION DURING DEVELOPMENT OF THE LABORATORY RAT. *(E.)* Foster, P. C.; Bailey, E. (Dep. Biochem., Univ. Sheffield, England). *Biochem. Soc. Trans.* 2(6):1279-1281, 1974.

1028 THE PHYSIOLOGICAL ROLE OF FATTY ACID ELONGATION IN PARTICULATE FRACTIONS OF RAT LIVER AND RELATIVE CONTROL FACTORS. *(E.)* Quagliariello, E.; Landriscina, C. (Dep. Biochem., Univ. Bari, Italy). *Biochem. Soc. Trans.* 2(6):1215-1218, 1974.

1029 METABOLIC ACTIVATION OF DRUGS TO TOXIC SUBSTANCES. *(E.)* Mitchell, J. R.; Jollows, D. J. (Natl. Heart Lung Inst., Bethesda, Md.). *Gastroenterology* 68(2):392-410, 1975.

1030 DEVELOPMENTAL CHANGE IN THE APPARENT KINETIC PROPERTIES OF DRUG-OXIDIZING ENZYMES IN THE HUMAN LIVER. *(E.)* Pelkonen, O. (Dep. Pharmacol., Univ. Oulu, Finland). *Res. Commun. Chem. Pathol. Pharmacol.* 10(2):293-302, 1975.

1031 PROTEIN DEFICIENCY IN CARBON TETRACHLORIDE-INDUCED HEPATIC LESIONS. *(E.)* Deo, M. G.; Roy, H.; Ramalingaswami, V. (All Indian Inst. Med. Sci., New Delhi). *Arch. Pathol.* 99(3):147-151, 1975.

1032 EFFECT OF PHENOBARBITAL AND CARBON TETRACHLORIDE ON DEHYDROCHOLATE INDUCED CHOLERESIS IN THE GUINEA-PIG. *(E.)* Chenderovitch, J.; Raizman, A.; Infante, R. (INSERM, St. Antoine Hosp., Paris, France). *Biol. Gastroenterol. (Paris)* 7(3):171-178, 1974.

1033 THE DIFFERENT SUSCEPTIBILITY OF RAT LIVER LOBES TO CARBON TETRACHLORIDE AND DIMETHYLNITROSAMINE. *(E.)* Lawson, T. A.; Pound, A. W. (Dep. Pathol., Univ. Queensland, Brisbane, Australia). *Br. J. Exp. Pathol.* 55(6):583-586, 1974.

1034 EFFECT OF CCl_4 ON KINETICS OF GALACTOSE ELIMINATION IN RAT LIVERS. *(E.)* Vilstrup, H.; Keiding, S.; Vendsborg, P. (Rigshosp., Copenhagen, Denmark). *Digestion* 10(4/5):384, 1974.

1035 CHARACTERISTIC CHANGES OF RAT LIVER LYSOSOMES IN ACUTE POISONING WITH CCl_4. *(Rus.)* Kondakova, A. E.; Korolenko, T. A.; Vakulin, G. M.; Shkurupii, V. A.; Iakobson, G. S. (Novosibirsk Med. Inst., USSR). *Tsitologiia* 15(11):1382-1389, 1973.

1036 THE ROLE OF LIVER PATHOLOGY IN OCCUPATIONAL DERMATOSES (EXPERIMENTAL STUDY). *(Rus.)* Mariasis, E. D.; Antoniev, A. A.; Prischepov, V. F.; Pavlik, L. V.; Makarov, V. N. (Med. Inst., Stavropol, USSR). *Gig. Tr. Prof. Zabol.* (9):17-21, 1974.

1037 ACUTE HEPATIC INJURY BY VINYL CHLORIDE IN RATS PRETREATED WITH PHENOBARBITAL. *(E.)* Jaeger, R. J.; Reynolds, E. S.; Conolly, R. B.; Moslen, M. T.; Szabo, S.; Murphy, S. D. (Kresge Cent. Environ. Health, Boston, Mass.). *Nature* 252 (5485):724-726, 1974.

1038 DRUG METABOLISM IN GUNN RATS: INABILITY TO INCREASE BILIRUBIN GLUCURONIDATION BY PHENOBARBITAL TREATMENT. *(E.)* Vainio, H.; Hietanen, E. (Dep. Physiol., Univ. Turku, Finland). *Biochem. Pharmacol.* 23(24):3405-3412, 1974.

1039 THE INFLUENCE OF BARBITURATES ON CHOLERESIS IS INDEPENDENT OF ENZYME INDUCTION. *(E.)* Capron, J. P.; Erlinger, S.; Feldmann, G.; Dumont, M. (INSERM, Beaujon Hosp., Clichy, France). *Digestion* 10(4/5):317-318, 1974.

1040 CHANGES IN THE PROTEIN-LIPID INTERACTION IN RAT LIVER MICROSOMES AFTER PRETREATMENT OF THE RAT WITH BARBITURATES AND POLYCYCLIC HYDROCARBONS. *(E.)* Laitinen, M.; Lang, M.; Hanninen, O.; (Dep. Physiol., Univ. Koupio, Finland). *Int. J. Biochem.* 5(9/10):747-751, 1974.

1041 HYDROXYLATION OF PHENACETIN BY BEAGLE LIVER MICROSOMES. *(E.)* Kampffmeyer, H. G. (Inst. Pharmacol., Univ. Munich, Germany). *Naunyn-Schmiedebergs Arch. Pharmacol.* 286(3):221-225, 1974.

1042 ALCOHOL AND DRUG METABOLISM. *(E.)* Nakanishi, S. (Sch. Med., Shinshu Univ., Matsumoto, Japan). *Asian Med. J.* 17(4):5-16, 1974.

1043 DISORDERS IN THE HEPATIC METABOLISM OF 1-^{14}C-PALMITATE PRODUCED BY ADMINISTRATION OF n-PROPANOL IN THE RAT. *(Fr.)* Beauge, F.; Clement, M.; Nordmann, J.; Nordmann, R. (Res. Group Intermed. Metab., INSERM, Paris, France). *Biochimie* 56(8):1157-1159, 1974.

1044 CHANGES OF MICROSOMAL STEROID-4en-5α-REDUCTASE ACTIVITY IN RAT LIVER BY ALCOHOL FEEDING. *(E.)* Bode, C.; Bode, J. C.; Martini, G. A. (Univ. Med. Clin., Marburg/Lahn, Germany). *Digestion* 10(4/5):337-338, 1974.

1045 ANIT CHOLESTASIS IN THE RAT: ALTERED LEVELS OF GLYCOLYTIC METABOLITES AFTER ETHANOL. *(E.)* Wolfert, W.; Ohnemus, H.; Goebell, H. (Dep. Intern. Med., Univ. Ulm, Germany). *Digestion* 10(4/5):388, 1974.

1046 HUMORAL AND CELL-MEDIATED IMMUNITY IN CHRONIC LIVER DISEASE. *(E.)* Thomas, H. C.; MacSween, R. N. M.; Tilney, N. L.; Horne, C. H. W. (Dep. Bacteriol., Immunol., Glasgow Univ., Scotland). *Digestion* 10(4/5):330, 1974.

1047 PHAGOCYTOSIS AND LYSOSOMAL DEGRADATION OF INTRAVENOUSLY INJECTED HUMAN-IGG IN THE MOUSE LIVER. *(E.)* Arnold, W.; Muller, O.; Mayersbach, H.; Meyer zum Buschenfelde, K. H. (2nd Dep. Intern. Med., Univ. Mainz, Germany). *Digestion* 10(4/5):335, 1974.

1048 STUDIES ON THE PATHOGENESIS OF THE EXPERIMENTAL ACTIVE CHRONIC HEPATITIS (ACH) IN RABBITS. *(E.)* Hopf, U.; Meyer zum Buschenfelde, K. H. (2nd Dep. Intern. Med., Univ. Mainz, Germany). *Digestion* 10(4/5):306-307, 1974.

1049 THE EFFECT OF PYRIDINOL CARBAMATE ON THE SYNTHESIS OF GLYCOSAMINOGLYCANS AND COLLAGEN IN CONNECTIVE TISSUE IN EXPERIMENTAL CIRRHOSIS IN RATS. *(Ger.)* Delbruck, A. (Oststadt Hosp., Hanover, Germany). *Z. Klin. Chem. Klin. Biochem.* 12(6):313-320, 1974.

1050 EFFECT OF FREE AND CONJUGATED CHENODEOXYCHOLATE ON LIVER CELL METABOLISM IN NORMAL AND BILE DUCT OBSTRUCTED PERFUSED RAT LIVERS. *(E.)* Liersch, M.; Hesse, W. (Med. Dep., Univ. Heidelberg, Germany). *Digestion* 10(4/5):364, 1974.

1051 INFLUENCE OF PREGNENOLONE-16α-CARBONITRILE ON THE LIVER ULTRASTRUCTURE IN ADRENALECTOMIZED, OVARIECTOMIZED, AND ORCHIDECTOMIZED RATS. *(E.)* Garg, B. D.; Szabo, S.; Tuchweber, B.; Kovacs, K. (Inst. Exp. Med. Surg., Montreal, Canada). *Acta Anat. (Basel)* 90(1):87-100, 1974.

1052 DOSE-DEPENDENT EFFECTS OF ETHINYLOESTRADIOL, DIETHYLSTILBOESTROL AND OESTRADIOL ON THE METABOLISM OF 4-ANDROSTENE-3,17-DIONE IN RAT LIVER MICROSOMES. *(E.)* Einarsson, K.; Gustafsson, J. A.; Sjovall, J.; Zietz, E. (Serafim Hosp., Stockholm, Sweden). *Acta Endocrinol. (Kbh.)* 78(1):54-64, 1975.

1053 INHIBITION OF ENZYMATIC REDUCTION OF Δ^{14}-DOUBLE BOND OF 5α-CHOLESTA-8,14-DIEN-3β-OL AND 5α-CHOLESTA-7,14-DIEN-3β-OL BY AY-9944. *(E.)* Lutsky, B. N.; Hsiung, H. M.; Schroepfer, G. J. (Sch. Chem. Sci., Univ. Illinois, Urbana). *Lipids* 10(1):9-11, 1974.

1054 EFFECT OF TRANS-1,4-BIS(2-CHLOROBENZYLAMINOMETHYL)-CYCLOHEXANE DIHYDROCHLORIDE AND CARBON MONOXIDE ON HEPATIC CHOLESTEROL BIOSYNTHESIS FROM 4,4-DIMETHYL STEROLS IN VITRO. *(E.)* Gibbons, G. F.; Mitropoulos, K. A. (Hammersmith Hosp., London, England). *Biochim. Biophys. Acta* 380(2):270-281, 1975.

1055 ALTERATIONS IN THE METABOLISM OF FATS AND PHOSPHATIDES IN RAT LIVER INDUCED BY HALIDE IONS. *(E.)* Leal, R. S.; Rodrigues, M. P. (Fac. Sci., Lisbon, Portugal). *Chem. Biol. Interact.* 10(2):91-101, 1975.

1056 IN VIVO HEPATIC AND INTESTINAL TOXICITY OF SODIUM CYANATE IN RATS: CYANATE-INDUCED ALTERATIONS IN HEPATIC GLYCOGEN METABOLISM. *(E.)* Haut, M. J.; Toskes, P. P.; Hildebrandt, P. K.; Glader, B. E.; Conrad, M. E. (Walter Reed Army Med. Cent., Washington, D.C.). *J. Lab. Clin. Med.* 85(1):140-154, 1975.

1057 THE DEPENDENCE ON VITAMIN E AND SELENIUM OF DRUG DEMETHYLATION IN RAT LIVER MICROSOMAL FRACTIONS. *(E.)* Giasuddin, A. S. M.; Caygill, C. P. J.; Diplock, A. T.; Jeffery, E. H. (R. Free Hosp. Sch. Med., London, England). *Biochem. J.* 146(2):339-350, 1975.

1058 CIRCADIAN OSCILLATION IN RAT LIVER TYROSINE AMINOTRANSFERASE ACTIVITY AFTER CHLOROFORM INHALATION. *(E.)* Stefan, M.; Gheorghie, N.; Boerescu, J. (D. Danielopolu Inst. Norm. Pathol. Physiol., Bucharest, Rumania). *Biochem. Pharmacol.* 24(4):479-480, 1975.

1059 ENZYME-HISTOCHEMICAL AND BIOCHEMICAL STUDY OF LIVER INJURY INDUCED BY LANTHANUM TRICHLORIDE. *(E.)* Kadas, I.; Tanka, D.; Keller, M.; Jobst, K. (Cty. Hosp., Pecs, Hungary). *Acta Morphol. Acad. Sci. Hung.* 22(1):35-45, 1974.

1060 FUNCTION AND MORPHOLOGY OF PIG'S LIVER INTOXICATED BY DADPM. *(E.)* Reiter, H. J.; Nitschke, J.; Schuster, H.; Laas, J.; Struck, E.; Hamelmann, H. (Surg. Clin., Phillips Univ., Marburg/Lahn, Germany). *Eur. Surg. Res.* 6(Suppl. 1):50-51, 1974.

1061 MORPHOLOGICAL RESULTS OF THE RAT LIVER AFTER THE APPLICATION OF HALOTHANE AND HYPOXIA. *(E.)* Leinweber, B.; Cuppers, M.; L'Allemand, H. (Med. Clin., Justus Liebig Univ., Giessen, Germany). *Acta Hepatogastroenterol. (Stuttg.)* 21(5):364-372, 1974.

1062 ALLOXAN INDUCED FATTY LIVER DEGENERATION IN RAT: ITS EFFECT ON THE MICROSOMAL LIPIDS. *(E.)* Hogberg, J. (Dep. Forensic Med., Karolinska Inst., Stockholm, Sweden). *Exp. Mol. Pathol.* 22(1):20-28, 1975.

1063 EFFECTS OF HYDROCORTISONE HEMISUCCINATE ON HEPATOCYTES FROM ADULT RAT LIVER IN MONOLAYER CULTURE. *(E.)* Gueguen, C.; Guillouzo, A.; le Cam, A.; Bourel, M. (INSERM, Pontchaillou Hosp., Rennes, France). *Digestion* 10(4/5):352-353, 1974.

1064 HEPATOCELLULAR ULTRASTRUCTURE FOLLOWING HYPERBARIC OXYGEN EXPOSURE. *(E.)* Balentine, J. D.; Greene, W. B. (Med. Univ. South Carolina, Charleston). *Am. J. Pathol.* 78(1):12a, 1974.

1065 ACUTE LIVER FAILURE BY ISCHEMIA AND INTOXICATION WITH MUSHROOM POISONS. *(E.)* Stoetter, L.; Erhardt, W.; Fischer, M.; Gartmaier, P.; Kircher, G.; Schwabe, M. (Dep. Surg., Isar Tech. Univ., Munich, Germany). *Digestion* 10(4/5):381, 1974.

1066 PROTECTIVE EFFECT OF AN ACTIVATED LIVERRES ON TOXIC LIVER CELL NECROSIS. *(E.)* Grun, M.; Liehr, H.; Rasenack, U. (Dep. Med., Univ. Wurzburg, Germany). *Digestion* 10(4/5):307, 1974.

1067 THE EFFECTS OF CATECHOLAMINES UPON THE METABOLISM OF FOREIGN COMPOUNDS AND UPON THE DISTRIBUTION OF PERFUSATE IN THE ISOLATED LIVER OF THE RAT. *(E.)* Boobis, A. R.; Powis, G. (Dep. Pharmacol., Glasgow Univ., Scotland). *Biochem. Pharmacol.* 23(24):3377-3384, 1974.

1068 INHIBITION OF SOME RAT HEPATIC MICROSOMAL ENZYMES BY ETHOXYQUIN. *(E.)* Parke, D. V.; Rahim, A.; Walker, R. (Dep. Biochem., Univ. Surrey, Guildford, England). *Biochem. Pharmacol.* 23(24): 3385-3394, 1974.

1069 THE EFFECT OF SEROTONIN ON LIVER FUNCTION IN ALBINO RATS. *(Rus.)* Olenik, A. N.; Venger, I. K.; Skakun, L. N.; Omelianiuk, L. S. (Ternopol Med. Inst., USSR). *Farmakol. Toksikol.* (37):186-188, 1974.

1070 EFFECT OF POLYENE PHOSPHATIDYLCHOLINE (EPL) ON EXPERIMENTAL LIVER INJURY. *(Jpn.)* Fujii, S.; Okuda, H.; Imagawa, T. (Dep. Enzyme Physiol., Univ. Tokushima, Japan). *Chikoka Acta Med.* 30(2):77-85, 1974.

1071 ULTRASTRUCTURAL CYTOCHEMICAL AND METABOLIC CHANGES AFTER NODULAR HYPERPLASIA IN EXPERIMENTAL HEPATOMEGALY. *(E.)* Robinson, N.; Nievel, J. G.; Anderson, J. (London Hosp. Med. Sch., England). *Biochem. Soc. Trans.* 2(6):1253-1254, 1974.

1072 PARTICIPATION OF VARIOUS HORMONES IN THE INDUCTION OF EXPERIMENTAL HEPATOMEGALY. *(E.)* Nievel, J. G. (King's Coll. Hosp. Med. Sch., London, England). *Biochem. Soc. Trans.* 2(6):1358-1359, 1974.

1073 PLASMINOGEN AND FIBRINOGEN SYNTHESIS IN EXPERIMENTAL HEPATOMEGALY. *(E.)* Scully, M. F.; Kakkar, V. V.; Nievel, J. G. (King's Coll. Hosp. Med. Sch., London, England). *Biochem. Soc. Trans.* 2(6):1254-1255, 1974.

1074 METABOLISM OF A CHOLERETIC AGENT, TRIMETHOXY-2'4,4'-CHALCONE (VESIDRYL). *(Fr.)* Chabannes, B.; Grandgeorge, M.; Duperray, B.; Pacheco, H. (Natl. Inst. Appl. Sci., Lyon, France). *Chim. Ther.* 8(6):621-625, 1973.

1075 ROLE OF VITAMIN A IN THE INDUCTION OF CYTOCHROME P-450 BY DDT. *(Fr.)* Ferrando, R.; Truhaut, R.; Graillot, C.; Gak, J. C.; Fourlon, C. (Natl. Vet. Sch., Alfort, France). *C. R. Acad. Sci. [D] (Paris)* 279(12):999-1001, 1974.

1076 THE EFFECT OF SELECTED COMPOUNDS ON THE ENZYMES IN RAT LIVER LYSOSOMES. I. IN VITRO INVESTIGATIONS. *(Pol.)* Ludwicki, J. K. (State Inst. Hyg., Warsaw, Poland). *Roczn. Panstw. Zakl. Hig.* 25(3):287-293, 1974.

1077 THE ANTIGENIC STRUCTURE OF PLASMA MEMBRANES OF THE MOUSE LIVER CELLS. I. ISOLATION OF TOTAL FRACTIONS OF LIPIDS AND PROTEIN IN THE LIVER CELL MEMBRANES AND EXAMINATION OF THEIR ANTIGENIC ACTIVITY. *(Rus.)* Khramkova, N. I.; Iakimenko, E. F.; Beloshapkina, T. D. (Inst. Epidemiol. Microbiol., Moscow, USSR). *Tsitologiia* 15(10):1284-1290, 1973.

1078 THE ISOLATED LIVER EFFICIENCY IN THE CLEARANCE OF VIABLE BACTERIA. *(E.)* Ruggiero, G.; Andreana, A.; Utili, R.; Giusti, G. (Univ. Naples 1st Med. Sch., Italy). *Digestion* 10(4/5):378, 1974.

1079 IMMUNIZATION OF MICE AGAINST THE TOXIC HEPATITIS PRODUCED BY FV_3: INHIBITION OF VIRUS PENETRATION INTO THE LIVER. *(E.)* Kirn, A.; Gut, J. P.; Steffan, A. M.; Gendrault, J. L. (INSERM, Louis Pasteur Univ., Strasbourg, France). *Intervirology* 2:244-252, 1973/74.

1080 HEPATIC ULTRASTRUCTURAL ABNORMALITIES IN EXPERIMENTAL CHRONIC RENAL FAILURE. *(E.)* Biempica, L.; Goldfischer, S.; Black, M.; Biempica, S. L.; Arias, I. M. (Albert Einstein Coll. Med., Yeshiva Univ., Bronx, N.Y.). *Am. J. Pathol.* 78(1): 13a, 1974.

1081 CONTROL OF KETOGENESIS FROM AMINO ACIDS. II. KETONE BODIES FORMATION FROM α-KETOISOCAPROATE, THE KETO-ANALOGUE OF LEUCINE, BY RAT LIVER MITOCHONDRIA. *(E.)* Noda, C.; Ichihara, A. (Sch. Med., Tokushima Univ., Japan). *J. Biochem.* 76(5):1123-1130, 1974.

1082 ON THE MECHANISM OF CUPRIZONE-INDUCED FORMATION OF MEGAMITOCHONDRIA IN MOUSE LIVER. *(E.)* Wakabayashi, T.; Green, D. E. (Inst. Enzyme Res., Univ. Wisconsin, Madison). *Bioenergetics* 6(5):179-192, 1974.

1083 ELECTRON MICROSCOPY AND MORPHOMETRY OF CANALICULAR DIFFERENTIATION IN FETAL AND NEONATAL RAT LIVER. *(E.)* De Wolf-Peeters, C.; De Vos, R.; Desmet, V.; Bianchi, L.; Rohr, H. P. (St. Rafael Acad. Hosp., Univ. Louvain, Belgium). *Exp. Mol. Pathol.* 21(3):339-350, 1974.

1084 SCANNING ELECTRON MICROSCOPY DEMONSTRATION OF CILIA IN RAT INTRAHEPATIC BILE DUCTS. *(E.)* Motta, P.; Fumagalli, G. (Fac. Med., Univ. Rome, Italy). *Z. Anat. Entwicklungsgesch.* 145(3): 223-226, 1974.

1085 BILE SALTS AND BILE FORMATION IN RATS, RABBITS AND GUINEA PIGS. *(E.)* Shaw, H. M.; Heath, T. J. (Sch. Physiol. Pharmacol., Univ. New South Wales, Kensington, Australia). *Comp. Biochem. Physiol.* [A] 50(3):615-619, 1975.

1086 THE CIRCADIAN RHYTHM OF BILE SECRETION IN THE RAT. *(E.)* Balabaud, C.; Noel, M.; Beraud, C.; Dangoumau, J. (Dep. Pharmacol., Univ. Bordeaux II, France). *Digestion* 10(4/5):325, 1974.

1087 THE METABOLISM OF TAURINE IN THE LIVING RAT. *(E.)* Federici, G.; Dupre, S.; Rosei, M. A.; Granata, F.; Orlando, M. (Inst. Biol. Chem., Univ. Rome, Italy). *Physiol. Chem. Phys.* 6(5):411-417, 1974.

1088 KINETIC STUDY OF LABELED BILIRUBIN. *(Fr.)* Berk, P. (Natl. Cancer Inst., Bethesda, Md.). *Ann. Gastroenterol. Hepatol. (Paris)* 10(3): 209-226, 1974.

1089 THE INFLUENCE OF TRUNCAL VAGOTOMY ON THE COMPOSITION OF BILE. *(E.)* Sier, J. C.; Visser, J.; Den Otter, G. (Inst. Exp. Surg., Free Univ., Amsterdam, Netherlands). *Eur. Surg. Res.* 6(1):50, 1974.

1090 ACTION OF THE CHRONIC IRRITATION OF THE SECTION OF THE VAGO-SYMPATHETIC NERVES UPON THE CHOLEDOCHAL SPHINCTER IN THE DOGS. *(E.)* Sarles, J. C.; Devaux, M. A.; N'Guyen, R.; Michel, G. (Dig. Pathol. Res. Unit, INSERM, Marseille, France). *Eur. Surg. Res.* 6(Suppl. 1):55-56, 1974.

1091 INDEPENDENCE FROM VAGAL CONTROL OF BILIARY SECRETION IN THE RAT. *(E.)* Debray, C.; de la Tour, J.; Roze, C.; Souchard, M.; Vaille, C. (Bichat Hosp., Paris, France). *Digestion* 10(6):413-422, 1974.

1092 ANGIOGRAPHIC STUDIES IN EXPERIMENTAL TOTAL AND LOBAR CHOLESTASIS IN DOGS. *(E.)* Bergan, A.; Enge, I.; Hall, C. (Inst. Surg. Res., Oslo, Norway). *Invest. Radiol.* 9(6):462-472, 1974.

1093 EXPERIMENTAL STUDIES FOR LITHOTRIPSY IN THE COMMON BILE DUCT. *(E.)* Deyhle, P.; Schauwecker, H.; Sauberli, H.; Largiader, F. (Surg. Canton. Hosp., Zurich, Switzerland). *Eur. Surg. Res.* 6(Suppl. 1):57, 1974.

1094 ABNORMAL LOW DENSITY LIPOPROTEIN IN PLASMA OF RAT WITH BILIARY OBSTRUCTION. A CHEMICAL AND MORPHOLOGICAL STUDY. *(E.)* Calandra, S.; Pasquali-Ronchetti, I.; Montaguti, M.; Cambi, M.; Caccone, P. (Inst. Gen. Pathol., Univ. Modena, Italy). *Digestion* 10(4/5):340, 1974.

1095 COMPLETE EXTRAHEPATIC OBSTRUCTIVE JAUNDICE--EXPERIMENTS ON DOGS. PART II. CHANGES IN BLOOD CLOTTING. *(Ger.)* Naber, K.; Muller, K.; Maroske, D.; Bichler, K.H.; Hupe, K.; Hoege, R. (Surg. Clin., Univ. Marburg/Lahn, Germany). *Bruns Beitr. Klin. Chir.* 221(1):55-69, 1974.

1096 EFFECTS OF CHOLERA TOXIN, GLUCAGON AND DIBUTYRYL CYCLIC AMP ON BILE FLOW AND BILE COMPOSITION. *(E.)* Baker, A. L.; Tse-Tu, D.; Kaplan, M. M. (Tufts-New England Med. Cent., Boston, Mass.). *Gastroenterology* 67(4):779, 1974.

1097 EFFECT OF BERBERINE ON BILE SECRETION. *(E.)* Oshiba, S.; Ueno, H.; Mihara, H.; Okamoto, S. (Nihon Univ. Sch. Med., Tokyo, Japan). *Nihon Univ. J. Med.* 16(1):69-79, 1974.

1098 THE BILIARY SECRETION OF RADIOACTIVE SELENIUM AFTER INTRAVENOUS INJECTION OF ^{75}Se-L-SELENO-METHIONINE. *(E.)* Pointner, H. (1st Dep. Med., Univ. Vienna, Austria). *Acta Hepatogastroenterol. (Stuttg.)* 21(5):380-382, 1974.

1099 EVIDENCE FOR A DOSE-DEPENDENT STIMULATION OR INHIBITION OF BILE FLOW BY CONTRAST MEDIA. *(E.)* Klapdor, R. (Med. Clin. I, Univ. Hamburg, Germany). *Digestion* 10(4/5):358-359, 1974.

1100 PHENOBARBITAL AND BILIARY EXCRETION OF CONTRAST MEDIA. *(E.)* Klapdor, R.; Valerius, H. (Med. Clin. I, Univ. Hamburg, Germany). *Digestion* 10(4/5):359, 1974.

1101 BILE SECRETION AND LIVER BLOOD FLOW DURING SECRETION INFUSION. *(E.)* Brunner, H.; Slat, B.; Kretschmer, G.; Funovics, J.; Grabner, G. (Surg. Clin. I, Univ. Vienna, Austria). *Digestion* 10(4/5):338-339, 1974.

1102 CONTRASTING EFFECTS OF DIFFERENT TYPES OF ENZYME INDUCERS OF CHOLESTEROL SATURATION OF RAT BILE. *(E.)* von Bergmann, K.; Schwarz, H. P.; Paumgartner, G. (Dep. Clin. Pharmacol., Univ. Bern, Switzerland). *Digestion* 10(4/5):317, 1974.

See also, 0811, 0819, 0820, 0825, 0837, 1118, 1122, 1126, 1320, 1376, 1461, 1592.

SECRETION AND METABOLISM

Intestine

1103 CANINE INTESTINAL SECRETION DURING AND AFTER RAPID DISTENTION OF THE SMALL BOWEL. *(E.)* Caren, J. F.; Meyer, J. H.; Grossman, M. I. (VA Cent., Los Angeles, Calif.). *Am. J. Physiol.* 227(1):183-188, 1974.

Intestinal secretion during and after rapid distention of the small bowel was measured in 4 mongrel dogs with extrinsically denervated Thiry-Vella loops of jejunum and ileum. Tactile stimulation of the mucosa by a condom deflated or slightly inflated (up to 15 ml) for 45 min significantly increased secretion during contact and for 30 min thereafter. This response was abolished by parenteral injections of atropine and hexamethonium although these drugs had no effect on intraluminal pressure. There was no increase in secretion during distention with 20 or 30 ml, but rises in fluid output were sustained for 2 or more hours after distention. This response was reduced by hexamethonium, but not atropine. Leakage of plasma protein, measured as the clearance of plasma-protein bound ^{51}Cr (injected i.v. at least 36 hr prior to distention) accounted for only 12% of secretions before, during, and after distention. Distention of ileum did not cause increased secretion in the jejunum, suggesting that a hormonal mechanism is not involved. Neither the mechanisms of the 2 patterns of secretory response nor the application to small-bowel obstruction can be deduced from this experiment.

1104 EFFECT OF VILLUS LENGTH ON CELL PROLIFERATION AND MIGRATION IN SMALL INTESTINAL EPITHELIUM. *(E.)* Rijke, R. P. C.; van der Meer-Fieggen, W.; Galjaard, H. (Med. Fac., Erasmus Univ., Rotterdam, Netherlands). *Cell Tissue Kinet.* 7(6): 577-586, 1974.

The life span and migration rate of epithelial cells on villi of experimentally reduced length in male Glaxo-Wistar rats was studied. Autoradiographic studies and scintillation counting were made at different time intervals after ^{3}H-thymidine labeling. The labeling was done at 36, 48, and 60 hr intervals after exposure to 700 r whole-body x-irradiation. The results of this study showed that the life span of epithelial cells in rat small intestine (36-48 hr) is independent of the villus length. In villi of reduced length the migration rate of the epithelial cells was decreased when compared to controls. Changes in the migration rate in turn seemed dependent on the production of epithelial cells in the crypt. Comparative studies on the recovery of crypt and villus epithelium after various doses (300 and 700 r) of x-irradiation support the theory that increased proliferative activity in the crypt cell compartment is related to a reduction of the number of functional villus cells below a critical villus length. The findings of this study are important in the interpretation of data on microbiochemical analyses of certain cell differentiation characteristics during periods of increased proliferative activity.

1105 ENZYME CHANGES IN HUMAN SMALL BOWEL MUCOSA DURING CULTURE IN VITRO. *(E.)* Mitchell, J. D.; Mitchell, J.; Peters, T. J. (R. Postgrad. Med. Sch., London, England). *Gut* 15(10):805-811, 1974.

Fluorimetric microassay methods were adapted to investigate enzyme activities in cultures of human jejunal biopsy tissue, and to develop a reproducible, quantitative means of assessing subcellular changes in intestinal mucosal tissue. Human jejunal biopsy slices from 5 normal adult volunteers (27-33 yr) and from one 22-yr-old male with Crohn's disease were maintained in culture (Eagle's basal medium) for up to 48 hr. At 24 hr there was good morphological preservation but by 48 hr there was ultrastructural evidence of damage to the enterocytes. During culture the tissue lost a certain amount of protein and the levels of 3 brush border enzymes (alkaline phosphatase, α-glucosidase, and leucyl-β-naphthylamidase) and 1 lysosomal enzyme (N-acetyl-β-glucosaminidase) showed a progressive decrease as determined by fluorimetric methods. Alkaline phosphatase, α-glucosidase, and N-acetyl-β-glucosaminidase accumulated in the medium throughout the experimental period to give a medium:tissue distribution ratio of between 2 and 9. Leucyl-β-naphthylamidase had a medium:tissue ratio of 140 after 48 hr of culture suggesting a selective secretion of this enzyme by the tissue. The reproducibility of the enzyme assays obtained indicates that detailed enzyme studies on pathological tissue can be undertaken.

1106 THE HYDROLYSIS OF PTEROYLPOLYGLUTAMATES IN THE SMALL INTESTINE. *(E.)* Jagerstad, M;

Westesson, A. K. (Res. Lab. Clin. Hematol., Univ. Lund, Sweden). *Scand. J. Gastroenterol.* 9(7):639-643, 1974.

Hydrolysis of synthetic pteroyloligoglutamates in the stomach and small intestine of male Wistar rats was studied by bioassay with *Lactobacillus casei* and *Streptococcus faecalis*. The rats were intubated with 0.1 μM of a preparation of synthetic pteroylglutamates containing either 1, 3, 4, or 5 glutamyl residues. After 30, 60, 90, and 120 min, the animals were anesthetized with ether, the stomach and small intestine removed and the small intestine divided into 3 parts. The contents from each intestinal part and the stomach were emptied, the folates extracted and the proteins precipitated. Folic acid concentrations assayed with both microorganisms were identical throughout the gastrointestinal tract. The pteroylpolyglutamates were partly hydrolyzed in the stomach and in the intestine up to the pancreatic duct. Thereafter the pteroylpolyglutamates occurred mainly in mono- and/or diglutamic form, as judged by the identical folate concentrations determined with *S. faecalis* and *L. casei*. Pteroylpolyglutamate hydrolase activity was found throughout the entire small intestine with the highest activity in the duodenum, jejunum and ileum; activity was also found in the area between the stomach and the pancreatic duct. No pteroylpolyglutamate-hydrolyzing activity was found in the stomach. It is suggested, therefore, that pteroylpolyglutamate hydrolase is secreted *via* the pancreatic juice.

1107 THE ROLE OF THE INTESTINAL MUCOSA IN CHOLESTEROL METABOLISM: ITS RELATION TO PLASMA AND LUMINAL CHOLESTEROL. *(E.)* Peng, S. K.; Ho, K. J.; Taylor, C. B. (VA Hosp., Albany, N.Y.). *Exp. Mol. Pathol.* 21(1):138-153, 1974.

Relationships between cholesterol in the intestinal mucosa, lumen, and plasma were studied in 3 groups of adult male albino rats: 13 fed a sterol-free diet, 13 fed a 2% cholesterol diet, and 7 fed a sterol-free diet followed by biliary diversion. Following intracardiac injection of cholesterol-4-^{14}C and sequential sacrifice, total cholesterol content and its radioactivity were measured in sera and in the mucosae and luminal contents of portions of the small intestine and colon. The cholesterol specific activity was greater in serum than mucosa and greater in the mucosa than in the lumen. This suggests 2 pools of cholesterol in the mucosa, 1 active in cholesterol synthesis and the other in total equilibrium with serum cholesterol. With sterol-free diet, 79% of the cholesterol in the active mucosa was synthesized *de novo* and contributed 77% of the fecal cholesterol. Transport from mucosa to lumen was mainly by active secretion and partly by desquamation. Mucosal cholesterol content, specific activity, and transport to the lumen were unaffected by cholesterol diet except for a 20% reduction of *de novo* synthesized cholesterol in the proximal small intestine. Biliary diversion caused a 5- to 6-fold increase of *de novo* synthesized cholesterol and a 2-fold increase in the active mucosal pool. These were attributed to the absence of bile salts which inhibit cholesterol synthesis and enhance absorption of cholesterol in the lacteals.

1108 EXPERIMENTAL ARTIFICIAL GUT BEFORE INTESTINAL TRANSPLANTATION IN DOG. *(E.)* Joyeux, H.; Astruc, B.; Yakoun, M.; Solassol, C. (Lab. Exp. Surg., Montpellier, France). *Eur. Surg. Res.* 6(Suppl. 1):53-54, 1974.

1109 FEATURES OF ENTEROCYTE POPULATION KINETICS IN VARIOUS SEGMENTS OF THE INTESTINAL TRACT AS A FACTOR CAUSING TUMOR DEVELOPMENT. *(Rus.)* Pozharisskii, K. M.; Klimashevskii, V. F.; Gushchin, V. A. (N. N. Petrov Sci. Res. Inst. Oncol., Leningrad, USSR). *Dokl. Akad. Nauk SSSR* 220(1):216-219, 1975.

1110 THE ROLE OF SUPERSENSITIVITY TO ACETYLCHOLINE IN THE PRODUCTION OF TOLERANCE TO MORPHINE IN STIMULATED GUINEA-PIG ILEUM. *(E.)* Shoham, S.; Weinstock, M. (Sackler Sch. Med., Tel Aviv Univ., Ramat Aviv, Israel). *Br. J. Pharmacol.* 52(4):597-603, 1974.

1111 PROTEIN DIGESTION DURING EARLY POSTNATAL DEVELOPMENT. *(Rus.)* Zufarov, K. A.; Tashkhodzhaev, P. I.; Gontmakher, V. M. (Tashkent Med. Inst., USSR). *Biull. Eksp. Biol. Med.* 77(10):12-15, 1974.

1112 EFFECT OF METHIONINE ON PEPTIDASE ACTIVITY OF THE CHICK SMALL INTESTINE. *(Rus.)* Kushak, R. I.; Sanzhur, T. S.; Fomina, G. A. (Inst. Biol., Riga, USSR). *Fiziol. Zh. SSSR* 60(11):1718-1722, 1974.

1113 EFFECT OF ADRENALECTOMY ON MUCOSAL ENZYME ACTIVITY IN THE RAT SMALL INTESTINE. *(Rus.)* Rakhimov, K.; Kadyrov, U. Z.; Zaripov, B. (Dep. Physiol., Acad. Sci. Uzbek SSR, Tashkent, USSR). *Biull. Eksp. Biol. Med.* 77(11):61-64, 1974.

1114 METABOLISM OF SERUM ALBUMIN IN EXPERIMENTAL INTESTINAL OBSTRUCTION. *(E.)* Herczeg, B. (Semmelweis Med. Univ., Budapest, Hungary). *Z. Exp. Chir.* 7(2):112-126, 1974.

1115 THE HYDROLYSIS OF STARCH AND SUCROSE BY THE RAT SMALL INTESTINE WITH IMPAIRED BLOOD SUPPLY AND LIGATED BILIARY AND PANCREATIC DUCTS. *(Rus.)* Pulatov, A. S.; Povetko, T. V. (M. I. Kalinin State Med. Inst., Andizhan, USSR). *Fiziol. Zh. SSSR* 60(11):1712-1717, 1974.

1116 AFFINITY LABELING OF SUCRASE-ISOMALTASE COMPLEX FROM RABBIT SMALL INTESTINE. *(E.)* Quaroni, A.; Di Capua, E.; Semenza, G. (Fed. Tech. Univ., Zurich, Switzerland). *Experientia* 30(6):690, 1974.

1117 THE INFLUENCE OF O-(β-HYDROXYETHYL)-RUTOSIDE (HR) ON INTESTINAL CELLS AFTER PARTIAL IRRADIATION. *(Ger.)* Oberhausen, V. H. (Inst. Biophys., Univ. Saarland, Homburg/Saar, Germany). *Arzneim. Forsch.* 25(1):101-105, 1975.

1118 IMMUNOCHEMICAL STUDY OF LARGE HUMAN INTESTINE ACID PHOSPHATASES. *(E.)* Benoliel, A. M.; Pianeta, C.; Sacoman, N.; Depieds, R. (Med. Teaching Res. Unit, Marseille, France). *Int. J. Biochem.* 5(7/8):667-672, 1974.

1119 SEASONAL CHANGES IN THE CIRCADIAN RHYTHMS OF MITOTIC ACTIVITY IN THE EPITHELIUM OF RECTAL CRYPTS IN RATS. *(Rus.)* Svetikova, K. M.; Katinas, G. S. (Inst. Exp. Med., Leningrad, USSR). *Arkh. Anat. Gistol. Embriol.* 67(7):62-68, 1974.

1120 THYROID CONTROL OF SMALL INTESTINAL OXYGEN CONSUMPTION AND THE INFLUENCE OF SODIUM IONS, OXYGEN TENSION, GLUCOSE AND ANAESTHESIA. *(E.)* Levin, R. J.; Syme, G. (Dep. Physiol., Univ. Sheffield, England). *J. Physiol. (Lond.)* 245(1):271-287, 1975.

See also, 0827, 0834, 0847, 0868, 1038, 1276.

GENERAL

1121 INTESTINAL VASOCONSTRICTION FOLLOWING DIURETIC-INDUCED VOLUME DEPLETION: ROLE OF ANGIOTENSIN AND VASOPRESSIN. *(E.)* McNeill, J. R. (Coll. Med., Univ. Saskatchewan, Saskatoon, Canada). *Can. J. Physiol. Pharmacol.* 52(4):829-839, 1974.

The i.v. administration of furosemide (1.0 or 2.0 mg/kg), ethacrynic acid (2.0 mg/kg) or chlorothiazide (20 mg/kg) induced intestinal vasoconstriction in pentobarbital-anesthetized (30 mg/kg sodium pentobarbital i.p.) cats (1.8-4.0 kg). The parameters in all animals were recorded for 30-60 min before and 90-180 min after administration of the agents. The intestinal vasoconstriction following furosemide was not reduced in animals subjected to acute intestinal denervation, adrenalectomy, and hypophysectomy. The response also was not reduced in animals subjected to intestinal denervation and adrenalectomy and given an inhibitor of angiotensin-converting enzyme, SQ20881 (Pyr-Trp-Pro-Arg-Pro-Gln-Ile-Pro-Pro; 1.0 mg/kg i.v.). However, administration of SQ20881 (1.0 mg/kg i.v. 30-40 min before administration of furosemide) to hypophysectomized animals completely abolished the vasoconstriction even when the intestinal innervation and adrenal glands remained intact. It is suggested that the renin-angiotensin system and the vasopressin system play major roles in the mechanism of intestinal vasoconstriciton following diuretic-induced volume depletion and that the sympathoadrenal system plays little role in this response.

1122 HEPATIC OUTFLOW RESISTANCE, SINUSOID PRESSURE, AND THE VASCULAR WATERFALL. *(E.)* Mitzner, W. (Dep. Biomed. Eng., Johns Hopkins Univ., Baltimore, Md.). *Am. J. Physiol.* 227(3):513-519, 1974.

The pressure-flow characteristics of the hepatic common outflow resistance were determined in 5 anesthetized (25 mg/kg pentobarbital) dogs. Portal venous pressure (P_{pv}) was measured *via* a catheter in the portal vein, and hepatic venous pressure (P_{hv}) was measured *via* a catheter in the left hepatic vein. A shunt containing an electromagnetic flow probe was established between the portal vein and abdominal inferior vena cava just downstream from the renal veins. Normal portal flow perfused the liver for 15 min. Hepatic artery flow was then set at 10 ml/min/kg and the portal flow was diverted into the inferior vena cava. Sinusoid pressure (P_s) was estimated as the pressure in portal vein with no portal inflow to the liver. The arterial flow was set to zero and flow rates increased in small steps. Two or 3 series of increasing-flow steps were performed on each animal and a pressure-flow curve determined. These curves were nearly linear in the range of normal flows, but exhibited a moderate convexity toward the pressure axis at low flows. The estimated (P_s) at zero hepatic flow was found to be higher than the outflow pressure. This closing pressure averaged 1.6 mm Hg relative to the P_{hv}. For an average P_{pv} of 7 mm Hg, P_{hv} of 1.6 mm Hg, and portal venous and hepatic arterial flows of 16.4 and 12.6 ml/min/kg, P_s was estimated to be 6.1 mm Hg. Two possible models (based on electric battery symbols) of the hepatic vasculature are presented. For the outflow resistance, one model assumes a single closing pressure, P_c, and a resistance R_L' which must decrease as P_s increases. The closing pressure is represented by a battery symbol of magnitude $P_c - P_{hv}$ and is in series with R_L'. As long as $P_{hv} < P_c$, there must exist a "vascular waterfall" of this magnitude. For $P_{hv} \geq P_c$, the magnitude of the waterfall is zero. The second model assumes a constant resistance, r_L, with a distribution of closing pressures providing an effective back pressure to flow of P_c'. This average closing pressure generates a vascular waterfall of mean magnitude $P_c' - P_{hv}$, for $P_{hv} < P_c$. For P_{hv} between P_c and the highest individual closing pressure in the bed, there will be a varying number of individual waterfalls, but the model does not hold. For P_{hv} greater than the highest individual closing pressure all waterfalls are zero.

1123 HEPATIC BLOOD FLOW IN THE RAT: EFFECT OF PORTACAVAL SHUNT. *(E.)* Ossenberg, F. W.; Denis, P.; Benhamou, J. P. (INSERM, Beaujon Hosp., Clichy, France). *J. Appl. Physiol.* 37(6):806-808, 1974.

The continuous BSP infusion method was used to assess hepatic blood flow in the rat. Blood from hepatic veins was sampled through a specially designed retrograde catheter. BSP extraction and hepatic blood flow were measured in 7 rats with end-to-side portacaval shunt and in 7 sham-operated rats 2 weeks after operation. BSP extraction was significantly lower in shunted rats than in sham-operated rats, 0.55 *versus* 0.68, resp. Hepatic blood flow/100 g was significantly lower in shunted rats than in sham-operated rats, 4.3 *versus* 7.8 ml/min, resp. As liver weight was lower in shunted than in sham-operated rats, 2.5 *versus* 3.7 g/100 g, resp., the hepatic blood flow/g liver was not significantly different in the two groups, 1.7 and 2.1 ml/min, resp. It was concluded from these data that the portacaval shunt induces a 35% decrease in hepatic blood flow when related to body weight, but does not change the mean value for hepatic blood flow when related to liver weight.

1124 THE CONFORMATIONS OF BILIRUBIN AND ITS ESTERS. *(E.)* Manitto, P.; Ricca, G. S.; Monti, D. (Inst. Org. Chem., Univ. Milan, Italy). *Gaz. Chim. Ital.* 104(7/8):633-637, 1974.

1125 PORTAL HYPOTENSION SAFELY MAINTAINED WITH VASOPRESSIN FOLLOWING HEMORRHAGE. *(E.)* Skivolocki, W. P.; Pace, III, W. G. (Ohio State Univ. Hosp., Columbus). *Eur. Surg. Res.* 6(Suppl. 1):3, 1974.

1126 THE IMMEDIATE EFFECTS OF HEPATIC ARTERY LIGATION ON LIVER HEMODYNAMICS AND LIVER FUNCTION IN THE CAT. *(E.)* Krarup, N.; Larsen, J. A. (Inst. Physiol., Univ. Aarhus, Denmark). *Acta Physiol. Scand.* 91(4):441-446, 1974.

1127 EVALUATION OF ^{14}C-TAUROCHOLATE FOR ESTIMATION OF HEPATIC BLOOD FLOW IN MAN. *(E.)* Paumgartner, G.; Reichen, J.; Preisig, R. (Dep. Clin. Pharmacol., Univ. Bern, Switzerland). *Digestion* 10(4/5):373, 1974.

1128 TRANSPLANTATION OF THE PANCREAS INTO A RETROPERITONEAL JEJUNAL LOOP. *(E.)* Dickerman, R. M.; Twiest, M. W.; Crudup, J. W.; Turcotte, J. G. (Univ. Michigan Med. Cent., Ann Arbor). *Am. J. Surg.* 129(1):48-54, 1975.

1129 CHANGES IN THE ABSORPTIVE CAPACITY OF THE SMALL INTESTINE DURING STEADY INCREASES IN PORTAL PRESSURE. *(Rus.)* Mozzhukhin, A. S.; Eremenko, V. P. (S. M. Kirov Mil. Med. Acad., Leningrad, USSR). *Biull. Eksp. Biol. Med.* 77(10):34-36, 1974.

1130 NUTRITIONAL FEATURES AND ECOLOGY OF PREDOMINANT ANAEROBIC BACTERIA OF THE INTESTINAL TRACT. *(E.)* Bryant, M. P. (Dep. Dairy Sci., Univ. Illinois, Urbana). *Am. J. Clin. Nutr.* 27(11):1313-1319, 1974.

1131 COMPOSITION OF INTESTINAL MICROFLORA IN MAN DURING QUALITATIVE AND QUANTITATIVE CHANGES OF PROTEIN IN THE DIET. *(Rus.)* Shilov, V. M.; Lizko, N. N.; Vysotskii, V. G.; Merkova, T. I. (Inst. Biomed. Probl., Moscow, USSR). *Zh. Mikrobiol. Epidemiol. Immunobiol.* (6):88-92, 1974.

1132 SIGNIFICANCE OF ANAEROBIC SPIROCHETES IN THE INTESTINES OF ANIMALS. *(E.)* Harris, D. L.; Kinyon, J. M. (Coll. Vet. Med., Iowa State Univ., Ames). *Am. J. Clin. Nutr.* 27(11):1297-1304, 1974.

1133 LACTOBACILLI WHICH ATTACH TO THE CROP EPITHELIUM OF THE FOWL. *(E.)* Fuller, R.; Brooker, B. E. (Natl. Inst. Res. Dairy., Shinfield, England). *Am. J. Clin. Nutr.* 27(11):1305-1312, 1974.

1134 STUDY OF ANTIGEN X WHICH IS RESPONSIBLE FOR PRODUCTION OF ANTI-Salmonella typhi ANTIBODIES, PROTECTORS OF THE CHICK EMBRYO. *(Fr.)* Joyeux, Y.; Jouin, H.; Leluc, B.; Staub, A. M. (Pasteur Inst., Paris, France). *C. R. Acad. Sci.* [*D*] *(Paris)* 279: 1507-1510, 1974.

See also, 0809, 0848.

DIAGNOSTIC PROCEDURES

1135 DEMONSTRATION OF SUPERFICIAL GASTRIC EROSIONS BY DOUBLE CONTRAST RADIOGRAPHY. *(E.)* Laufer, I.; Hamilton, J.; Mullens, J. E. (McMaster Univ. Med. Cent., Hamilton, Canada). *Gastroenterology* 68(2):387-391, 1975.

Radiological and endoscopic diagnosis of superficial gastric erosions was reviewed to show the value of double contrast radiography in the diagnosis of these small lesions. Endoscopic examinations were conducted on 267 patients, in 30 of whom such erosions were reported. In 12 patients there were coexisting peptic ulcers of the stomach or duodenum, while in the remainder the erosions were isolated abnormalities. Predisposing factors in 14 patients included acetylsalicylic acid (5), ethanol (4), gastric surgery (2), nongastric surgery (1), prednisone (1) and Crohn's disease (1). Four patients presented with gastrointestinal bleeding, the rest complained of dyspepsia or epigastric pain often like peptic ulcer pain. Twenty-five of the patients were examined by the double contrast method, and superficial gastric erosions were demonstrated radiologically in 13 of them and in 7 others not submitted to endoscopy. The erosions appeared as flat, linear streaks, as dots of barium, or as target-like lesions with a central fleck of barium surrounded by a radiolucent halo. In one patient, a ring-shaped density was demonstrated due to a small blood clot adherent to a gastric erosion. Although patients presenting with acute hemorrhagic gastritis should be examined first by endoscopy, double contrast radiography has been of value in patients presenting after the cessation of bleeding and in patients with vague digestive complaints. It is suggested that this type of examination be applied more widely and that it may eventually become the standard technique for radiological examination of the upper gastrointestinal tract.

1136 INCREASED SENSITIVITY OF THE CHOLYLGLYCINE BREATH TEST FOR DETECTING ILEAL DYSFUNCTION. *(E.)* Hepner, G. W. (Milton S. Hershey Med. Cent., Hershey, Pa.). *Gastroenterology* 68(1):8-16, 1975.

Deconjugation of bile salts was assessed by measuring the rate of appearance of $^{14}CO_2$ in the breath after administering cholyl(1-^{14}C)glycine to 19 patients with ileal dysfunction (6 men and 3 women mean age 36 yr with ileitis, plus 7 men and 3 women mean age 40 yr with ileal resection), 23 patients with cholesterol cholelithiasis (17 women and 6 men mean age 36 yr) with or without cholecystectomy, and 30 control subjects either healthy (7 men and 7 women mean age 31 yr) or with diarrhea (10 women and 6 men mean age 51 yr). Vitamin B_{12} absorption was also measured in many with ileal dysfunction by determining the recovery of ^{57}Co in the urine (Schilling test). The percentage of administered ^{14}C excreted in breath $^{14}CO_2$ was increased in ileal dysfunction patients, but there was considerable overlap between those with ileal dysfunction and controls. Breath $^{14}CO_2$ was excreted more rapidly in all patients with ileal dysfunction than in controls. Of the calculated 24-hr breath excretion, 78.3% of administered ^{14}C was excreted in 12 hr in ileal dysfunction patients, compared to 39.1% in controls. Only 1 of the 19 patients with ileal dysfunction overlapped the controls. The Schilling test was normal or equivocal in 5 of 14 patients with ileal dysfunction. The percentage of administered ^{14}C excreted in breath $^{14}CO_2$ was increased in patients with cholecystectomy, but excretion was not more rapid than in controls. The data suggest that the sensitivity of the breath test for diagnosing ileal dysfunction may be usefully increased by determining the rate of excretion of breath $^{14}CO_2$. These data further suggest that the increased exposure of the bile acid pool to deconjugating bacteria in patients with cholesterol cholelithiasis and cholecystectomy is small compared with that seen in ileal dysfunction patients, and there is no increased fecal loss of ^{14}C from cholyl (1-^{14}C)glycine in these patients.

1137 CHANGES IN SERUM PHOSPHATE LEVELS ASSOCIATED WITH INTESTINAL INFARCTION AND NECROSIS. *(E.)* Jamieson, W. G.; Lozon, A.; Durand, D.; Wall, W. (no affil.). *Surg. Gynecol. Obstet.* 140(1):19-21, 1975.

Serum phosphate determinations were made in 7 patients with massive intestinal infarction. The phosphate levels were elevated in each of the 7, 6 of whom had mesenteric vascular occlusion and 1 had midgut volvulus with intestinal necrosis. Experimental ligation and division at the origin of superior mesenteric artery was carried out in 25 mongrel dogs while an additional 15 underwent ligation and division of the superior mesenteric vein at its junction with the portal vein. The dogs had bloody diarrhea at 4 to 6 hr after operation. They were lethargic and febrile. Inorganic phosphate levels were significantly elevated in all dogs having ligation of the superior mesenteric artery. The phosphate levels were less elevated in those undergoing venous ligation. Hemoglobin and hematocrit levels were also elevated. Portal venous blood showed marked elevation of inorganic phosphate after ligation of the superior mesenteric artery. A significant metabolic acidosis with base deficit occurred, probably due to flooding of the systemic circulation with inorganic phosphate ion. Autopsy confirmed intestinal infarction in all dogs. Bioassay showed significantly higher levels of inorganic phosphate in the jejunum compared with other tissues. It was concluded that the serum phosphate level is increased in patients with massive intestinal necrosis. If only a small area of intestine is necrotic, however, the peripheral vein serum phosphate levels do not rise and the released phosphate fails to escape the liver. It is suggested that elevated serum phosphate levels, metabolic acidosis with significant base deficit, elevated WBC, and elevated hemoglobin and hematocrit values, when coupled with pertinent clinical signs and symptoms, may indicate the need for laparotomy and thus shorten the time period between vascular occlusion and operation.

1138 CHOLECYSTOKININ CHOLANGIOGRAPHY AND ANALYSIS OF DUODENAL BILE IN THE INVESTIGATION OF PAIN IN THE RIGHT UPPER QUADRANT OF THE ABDOMEN WITHOUT GALLSTONES. *(E.)* Freeman, J. B.; Cohen, W. N.; DenBesten, L. (Univ. Iowa Hosp. Clin.,

Iowa City). *Surg. Gynecol. Obstet.* 140(3):371-376, 1975.

A prospective study was carried out to test the hypothesis that the combined use of preoperative cholecystokinin cholangiography, duodenal drainage, and analysis of biliary lipids will enable the selection of those who would benefit from laparotomy. Patients (31; 29 female) with persistent biliary tract symptoms and at least two normal oral cholecystograms were studied. The symptoms consisted of pain in the epigastric or right upper quadrant region, frequently with radiation to the right scapula. Based on combined cholecystokinin cholangiography and biliary drainage studies, cholecystectomy was performed in 22 patients. The indication for surgery in 10 cases was a reduplication of clinical symptoms with pain lasting for at least 10 min after cholecystokinin injection. The symptoms were caused by resistance to gallbladder emptying, secondary to dyscoordinate contractions at the neck of the gallbladder or to obstruction of the cystic duct. In seven other patients, there was a virtual absence of gallbladder contractility, without pain, following cholecystokinin injection. The symptoms were due to hypokinetic contractions, and no patient had occlusion of the cystic duct. In five additional patients, there was neither pain nor delayed emptying of the gallbladder after cholecystokinin injection. Each of these patients had supersaturated duodenal bile with crystals and/or bacteria. Twenty patients recovered completely following surgery, and the other two showed definite improvement; there were no therapeutic failures. Cholecystokinin cholangiography quickly and capably detects the presence of neuromuscular disease of the gallbladder wall, and, combined with microscopic and bacteriologic examination of the duodenal bile, has great potential benefit for the patient and clinician.

1139 LIMITATIONS OF THE USEFULNESS OF THE d-XYLOSE ABSORPTION TEST. *(E.)* Krawitt, E. L.; Beeken, W. L. (Dep. Med., Univ. Vermont, Burlington). *Am. J. Clin. Pathol.* 63(2):261-263, 1975.

The results of the conventional 5-hr *d*-xylose absorption test were surveyed in 38 subjects with disease of the jejunal mucosa (including celiac sprue, Whipple's disease, intestinal lymphangiectasia and lymphoma), giardiasis, or small-intestinal bacterial overgrowth. None of the patients had renal dysfunction or neurologic defects. All were hydrated and had adequate urinary volumes. Although the results of *d*-xylose excretion tests of patients tended to be abnormal, and those of controls generally fell into the normal range, there was marked overlap in all groups. Two patients with severe celiac sprue showed normal *d*-xylose excretion values. These data indicate that the *d*-xylose test as it is conventionally performed results in false-positives and false-negatives in about 30% of the subjects tested, regardless of diagnosis. Thus, in any individual case it has limited usefulness in providing meaningful data in either identifying malabsorption or leading to a small-bowel biopsy, a search for the presence of giardiasis, or a quantitative small-bowel culture. It is concluded that there is no reason to perform the conventional *d*-xylose absorption test in the work-up of patients when jejunal biopsies can be easily and safely obtained.

1140 CARCINOMA OF THE PANCREAS: DETECTION AND MONITORING BY CARCINOEMBRYONIC ANTIGEN (CEA) AND ULTRASONOGRAPHY. *(E.)* Lokich, J. J.; Chawla, P. L.; Smith, E. H.; Zamcheck, N. (Harvard Med. Sch., Boston, Mass.). *Am. J. Gastroenterol.* 62(6):481-487, 1974.

Twenty-three patients (15 men and 8 women, aged 40-84 yr) with pancreatic carcinoma were followed sequentially by serial plasma carcinoembryonic antigen (CEA) determinations and abdominal ultrasonography. Five patients with normal CEA levels (less than 2.5 ng/ml) survived from less than 3 months to more than 12 months, median 8 months. CEA levels were elevated in 17 patients (74%). With hepatic metastasis, CEA levels ranges from 2.5 to 45 ng/ml, with 50% of these falling in the 2.5 to 15 ng/ml range. For those with levels up to 10 ng/ml, survival was similar to patients with normal CEA levels. Patients with hepatic metastasis and levels of CEA below 15 ng/ml had a longer survival which was not related to quantitative differences in tumor bulk. Of the seven patients with initial CEA levels over 10 ng/ml, all survived less than 6 months, and 3 less than 3 months, median 4 months. Thus, high CEA levels of over 10 ng/ml are generally indicative of a poor prognosis while levels less than 10 ng/ml indicate longer median survival. A direct correlation of serial CEA levels with clinical disease activity was established in a majority of the 10 cases in which it was investigated. A rising CEA level in increments of more than 3 ng/ml indicated the onset of metastatic disease, particularly for those patients with initial CEA levels of less than 15 ng/ml. A specific correlation of CEA levels with regressive disease was possible in only 1 patient. Ultrasonography was performed in 12 patients with pancreatic cancer. In all but 1 patient detection of a mass lesion was possible. Five patients had multiple ultrasound examinations during the course of the disease to monitor response to chemotherapy. No clinical response to therapy was observed, and in all 5 progressive enlargement of pancreatic mass lesions or retroperitoneal disease was determined by ultrasound. In general, the ultrasound could detect the lack of tumor response prior to clinical deterioration. Sequential ultrasound examinations were related to CEA levels to provide evidence of recurrent disease in 1 case, thus allowing a change in therapeutic regimen to be instituted earlier. The combined use of CEA determinations and ultrasonography to monitor patients with pancreatic carcinoma is useful in clinical management. The major advantages of these techniques are their simplicity and the direct quantitation of tumor mass.

1141 A COMPARISON BETWEEN SECRETIN ALONE AND SEQUENTIAL AND SIMULTANEOUS SECRETIN AND CHOLECYSTOKININ ADMINISTRATION IN THE ASSESSMENT OF PANCREATIC FUNCTION. *(E.)* Clain, J.; Bank, S.; Barbezat, G. O.; Novis, B. H.; Marks, I. N. (Groote Schuur

Hosp., Cape Town, South Africa). *Gut* 15(11):885-888, 1974.

A study was undertaken to compare sequential and simultaneous hormone administration, and to establish whether secretin alone was comparable to secretin combined with cholecystokinin as a stimulus to pancreatic secretion. Twenty-five patients with suspected pancreatic disease participated in the study. Each patient underwent 2, 80-min pancreatic function tests, in tandem, the first separated from the second by 30 min. For these tests submaximal doses of the hormones were administered by slow i.v. infusion. Five patients received secretin followed 60 min later by cholecystokinin for both tests. A second group of 5 patients received secretin followed 60 min later by cholecystokinin for the first test, and received secretin and cholecystokinin simultaneously in the second test. This order of the tests was reversed in a third group of 5 patients. In a fourth group, secretin was given first, followed by cholecystokinin at 60 min for the first test, and cholecystokinin was given first followed by secretin at 20 min for the second test. In a final group of 5 patients, this order of testing was reversed. Results of these studies indicated that volume, bicarbonate concentration, and enzyme secretion were the same whether secretin was followed by cholecystokinin or cholecystokinin followed by secretin or the two hormones were given simultaneously. When comparing patients who received a 60-min secretin stimulation alone with those who received a 60-min combined stimulation with secretin and cholecystokinin, the mean volume output and bicarbonate concentrations were similar, but the addition of cholecystokinin affected the mean enzyme concentration. This concentration was higher for all 4 enzymes in the test, although only significantly so for trypsin. It was concluded that cholecystokinin increases the concentration of enzymes in the pancreatic juice, but that its time of administration in relation to secretin is not critical in the assessment of pancreatic function.

1142 SPECIFICITY OF INCREASED RENAL CLEARANCE OF AMYLASE IN DIAGNOSIS OF ACUTE PANCREATITIS. *(E.)* Warshaw, A. L.; Fuller, Jr., A. F. (Massachusetts Gen. Hosp., Boston). *N. Engl. J. Med.* 292(7):325-328, 1975.

The amylase:creatinine clearance ratio was calculated in 86 patients with hyperamylasemia. These patients had acute pancreatitis (32 cases); common-duct obstruction with (10) or without pancreatitis (11); benign peptic ulcer of the stomach or duodenum (9); salivary adenitis (4); intestinal obstruction (5); intestinal ischemia (2); and regional enteritis (2); macromaylasemia (3); diabetic ketoacidosis (1); renal failure (1); liver trauma (3); hepatitis (2); and perforated duodenal ulcer (1). In this patient population the distribution of amylase values was similar whether patients had acute pancreatitis or another disease. The 3 patients with the highest levels of serum amylase did not have pancreatitis. The amylase:creatinine clearance in controls averaged 3.1%. In acute pancreatitis with or without an associated common-duct stone, only 3 of 42 patients had a clearance ratio below 5.3%. In contrast, the amylase:creatinine clearance ratio remained within the normal range in 44 patients with hyperamylasemia due to disease other than pancreatitis. The ratio of urinary amylase concentration to urinary creatinine concentration rose markedly in acute pancreatitis, but similar rises occurred in patients with hyperamylasemia resulting from other diseases. Serum lipase did not distinguish pancreatitis patients with or without common stones from patients with stones but without pancreatitis. These results suggest that renal permeability to amylase is altered in acute pancreatitis, and provides a basis for using the amylase:creatinine clearance ratio in the differential diagnosis of hyperamylasemia.

1143 PLASMA ALKALINE PHOSPHATASE ISOENZYMES IN HEPATOBILIARY DISEASE. *(E.)* Afonja, A. O.; Baron, D. N. (R. Free Hosp., London, England). *J. Clin. Pathol.* 27(11):916-920, 1974.

Plasma alkaline phosphatase isoenzyme patterns were investigated by cellulose acetate or acrylamide gel electrophoresis in a variety of hepatobiliary disorders and in some other conditions for comparison. Normal sera were obtained from fasting laboratory staff and from children with unrelated complaints. Abnormal sera were obtained from patients with hepatobiliary disorders. The sera were analyzed first for total alkaline phosphatase activity by the kinetic method to determine the amount of serum needed for electrophoresis. On cellulose acetate, 6 µl of serum containing less than 20 King-Armstrong (KA) U/dl was used, while 3 µl was sufficient for serum containing higher activities. On acrylamide gel, 25 µl of serum containing less than 30 KA U/dl was used; when 30-60 KA U/dl, 20 µl; 60-90 KA U/dl, 15 µl; 90-120 KA U/dl, 10 µl; 120-150 KA U/dl, 5 µl. The serum was diluted with aqueous sucrose solution (20 g/dl) if the activity exceeded 150 KA U. Since the heat-stable placental and Regan isoenzymes have a mobility similar to but distinguishable from the bone isoenzyme, their presence was confirmed by repeating the electrophoretic separation after destroying the bone isoenzyme by heat. Differentiation of intestinal from liver isoenzyme was confirmed by its sensitivity to 5 mM/liter of L-phenylalanine, while bone and liver isoenzymes were characterized by their sensitivity to 25 mM/liter of L-homoarginine. Acrylamide electrophoresis was performed at 4 C for 6 hr at 90 V, and cellulose acetate electrophoresis at room temperature for 18 min at 200 V. The order of separation of isoenzymes from sera from typical clinical cases is as follows: (anode) fast liver, slow liver, placental Regan, bone, intestinal, bile (origin): (cathode). The acrylamide gel method separated the bone and 'slow' liver isoenzymes more clearly but the bile isoenzyme could not be identified with certainty. Results were otherwise identical with either method. Normal adult serum always contained slow-liver isoenzyme, and sometimes bone isoenzyme; the latter was always present in the serum of children. In hepatobiliary disease slow-liver isoenzyme was always increased; intestinal isoenzyme appeared in many cases of cirrhosis (blood groups B

and 0) but fast-liver and bile isoenzymes were occasionally seen in miscellaneous cases. Regan isoenzyme was found in 6 of 45 cases of malignant disease. It is concluded that the ease of setting up and the short time required for adequate electrophoretic separation make the cellulose acetate strip a suitable method for the routine analysis of alkaline phosphatase isoenzymes in the serum.

1144 GAMMA-GLUTAMYL TRANSPEPTIDASE: CHOLESTATIC OR CYTOLYTIC ENZYME? *(Fr.)* Bertrand, L.; Arnefaux, J. (St. Eloi Hosp., Montpellier, France). *J. Med. Lyon* 54(1240):29-35, 1973.

To ascertain their relative value in diagnosis and follow-up, comparisons were made of serum γ-glutamyl transpeptidase (GGT), total bilirubin, alkaline phosphatase, and SGOT in 948 patients with a variety of liver and other diseases. GGT values were moderately increased in 63 patients with viral hepatitis, 3 with primary biliary cirrhosis, and 29 with alcoholic steatosis; SGOT was more useful in the diagnosis and follow-up of viral hepatitis. Much higher values of GGT were found in 10 patients with alcoholic necrosis and in 32 with obstructions of the extrahepatic biliary tract. Very high values (about 440 U/liter) were found in patients with incomplete obstruction due to choledocholithiasis (14 cases) or stenosis of the sphincter of Oddi (3 cases), while somewhat lower values (about 320 U/liter) were present in 15 patients with complete biliary obstructions caused by tumors in the head of the pancreas. Alkaline phosphatase values were similarly increased, but to a lesser extent. Surgical removal of the obstruction was followed by a rapid decrease in GGT values. Very large increases in GGT were also found in 35 patients with liver cancer: these values were higher among 19 patients with hepatic metastases than among 16 with primary liver cancer. Because GGT values gradually increase, they are valuable for early diagnosis of hepatic metastases. No increase occurred in GGT in 14 patients with cholestasis of pregnancy although alkaline phosphatase values doubled. On the basis of these findings and experimental findings reported in the literature, it is concluded that GGT is an indicator of hepatic necrosis rather than of cholestasis or cytolysis.

1145 SCAN EVIDENCE OF DECREASE IN SIZE OF INTRAHEPATIC TUMORS AFTER CHEMOTHERAPY. A CASE REPORT. *(E.)* Witek, J. T.; Spencer, R. P. (Yale Univ. Sch. Med., New Haven, Conn.). *Gastroenterology* 67(3):516-518, 1974.

Three cases are presented in which there was scan-documented metastatic tumor in the liver. A 38-yr-old woman showed, at laparotomy, an extensive carcinoma of the cecum. A mass, present in the right lobe of the liver, proved to be a metastatic carcinoma. The patient was started on 5-fluorouracil, and a repeat scintiscan obtained 49 days after the first study showed a decrease in the size of the lesion. A liver scintophoto in a 70-yr-old man who had been seen 7 yr previously for resection of carcinoma of the rectosigmoid, revealed an organ 19 cm in length, with at least 1 large defect in the upper portion of the right lobe. Therapy with 5-fluorouracil was begun and a repeat scintiscan, obtained 82 days after the first study, revealed almost complete disappearance of the defect. A 52-yr-old man had a nephrectomy for transitional cell carcinoma of the left kidney and 3 months later a liver scintophoto showed several defects. A course of adriamycin was administered at monthly intervals for a total of 6 courses. A repeat liver scintophoto 11 months after the initial study revealed almost complete reversion to normal. It is concluded that liver scans can provide one of the few objective criteria of the response to chemotherapy.

1146 A COMPARISON OF THE ACCURACY OF PERITONEOSCOPY AND LIVER BIOPSY IN THE DIAGNOSIS OF CIRRHOSIS. *(E.)* Bruguera, M.; Bordas, J. M.; Mas, P.; Rodes, J. (Prov. Hosp. Clin., Univ. Barcelona, Spain). *Gut* 15(10):799-800, 1974.

The accuracy of peritoneoscopy and liver biopsy in the diagnosis of hepatic cirrhosis was compared in 473 consecutive patients submitted to both procedures. Cirrhosis was diagnosed by one or both methods in 152 of these patients. There was 73% agreement (111 cases) between the two procedures. Of the 41 cases in which a discrepancy existed, cirrhosis was diagnosed histologically in 27 and peritoneoscopically in 14. The apparent false-negatives were 17.7% for peritoneoscopy and 9.3% for liver biopsy. The etiology of the 152 cases of cirrhosis were: cryptogenic 83, alcoholic 64, Wilson's disease 2, and 1 each for idiopathic hemochromatosis, Thorotrast, and secondary biliary cirrhosis. Of the 27 patients diagnosed incorrectly by liver biopsy, 26 had hepatic fibrosis and 1 had a normal liver. Of the 14 patients diagnosed incorrectly by peritoneoscopy, 3 had chronic aggressive hepatitis, 2 alcoholic hepatitis, 1 submassive hepatic necrosis, 1 normal liver, 1 nonspecific reactive hepatitis and 6 were not diagnostic. Although this study showed a high accuracy for peritoneoscopy and liver biopsy singly, 82.3% and 90.7%, resp., combining the methods afforded a significant increase in positive results. It is suggested that combined peritoneoscopy and biopsy methods be incorporated into the diagnostic procedure for suspected cirrhosis.

1147 ANGIOGRAPHIC FINDINGS OF CHOLEDOCHAL CYST. *(E.)* Jacobs, R. P.; Palubinskas, A. J. (Univ. California Sch. Med., San Francisco). *Br. J. Radiol.* 48:51-52, 1975.

An 18-yr-old girl presented for evaluation of a mass in the right upper quadrant of the abdomen which she had noted 10 days earlier. The mass was large, soft, non-tender and distinct from the liver. Laboratory studies indicated biliary obstruction: bilirubin was 3.1 mg/dl, alkaline phosphatase 319 IU/liter. Excretory urography showed a large mass with cephalad displacement of the right kidney with stretching of the right ureter. Barium enema showed depression of the hepatic flexure and anterior displacement of the proximal aspect of the transverse colon. Upper gas-

trointestinal tract examination showed slight displacement of the stomach to the left and marked anterior displacement, stretching and effacement of the descending duodenum. A liver scintiphoto study showed a normal sized liver with 2 large, central zones of decreased activity. At this stage the working diagnosis was abdominal malignancy with hepatic metastasis. Celiac arteriograms showed the mass was cystic, stretching the gastroduodenal artery and its branches anteriorly and elevating the right hepatic and cystic arteries. Superior mesenteric arteriography showed displacement of the inferior pancreaticoduodenal, the right colic, and the ileocolic branches about the cyst. A preoperative diagnosis of choledochal cyst was made by these angiographic findings: retroduodenal cyst of right upper quadrant; normal cystic artery without evidence of gallbladder distension; no angiographic evidence of malignancy; and central, tubular defects in the hepatogram phase. This may be the first case in which diagnosis was made by angiography.

1148 GALL-BLADDER EMPTYING MEASURED BY A RADIOISOTOPIC METHOD. *(E.)* Chapple, M. J.; Nolan, D. J.; Low-Beer, T. S.; Davies, E. R. (United Bristol Hosp., England). *Br. J. Radiol.* 48(565):19-22, 1975.

Oral radioisotope cholecystography was used to measure gallbladder emptying in 34 patients. In order to test the principle of the method a model was constructed using a balloon immersed in a water bath. Each of the 34 patients was being tested for suspected gallbladder disease. On the evening before examination the patient took 3 g sodium ipodate with a small volume of 10 µCi ^{131}I sodium ipodate in solution. The next morning the standard examination of the gallbladder was conducted. Gallbladder films were taken to measure gallbladder volume and localize the surface markings of the gallbladder. A collimated scintillation counter was suspended vertically over the area of maximal radioactivity. The recordings began immediately before ingestion of an emulsion of 20 ml arachis oil and continued until contraction of the gallbladder was maximal. Another radiograph in the supine position was taken at the end of the examination. The results of the study indicate that the isotope cholecystogram is a satisfactory method of monitoring emptying of the contracting gallbladder. Its results correlate well with changes in volume measured radiographically and it has the advantage of showing the pattern of contraction as well as the degree. Its ease and low radiation hazard make it a useful technique for studying gallbladder function. If combined with cholecystokinin assay, it may be useful in investigating the relation between gallbladder malfunction and disorders of intestinal function which may be associated with abnormalities of cholecystokinin release.

1149 SERUM LYSOZYME IN CROHN'S DISEASE AND ULCERATIVE COLITIS. *(E.)* Falchuk, K. R.; Perrotto, J. L.; Isselbacher, K. J. (Harvard Med. Sch., Boston, Mass.). *N. Engl. J. Med.* 292(8):395-397, 1975.

Serum lysozyme (muramidase) concentrations were determined in patients with different types of inflammatory bowel disease and in normal subjects. The patients included 20 with ulcerative colitis (13 males and 7 females aged 10-73 yr); 25 with Crohn's disease (12 males and 13 females aged 12-78 yr); 2 with salmonella enterocolitis (aged 18 and 24 yr); and 4 with nonbacterial enteritis (19-34 yr). The mean lysozyme concentration for each group was as follows: controls, 8.8 µg/ml; ulcerative colitis, 9.3; Crohn's disease, 26.3; and bacterial or nonbacterial enteritis, 8.9. The mean enzyme level for Crohn's disease patients was significantly greater than for other groups. Also, there was no overlap of the serum lysozyme concentration between patients with Crohn's disease and those with ulcerative colitis. In Crohn's disease, no correlation was found between the serum lysozyme concentration and the extent of disease. The highest serum lysozyme concentrations in patients with ulcerative colitis were found in a patient with toxic megacolon and a patient with severe ulcerative colitis in whom peritonitis secondary to a colonic perforation developed. No correlation was noted between the serum lysozyme concentration and total WBC. Neither was there a correlation between serum lysozyme and the absolute number of circulating granulocytes and monocytes. The elevation of serum lysozyme in Crohn's disease may be related to tissue macrophages. It is concluded that an increased level of serum lysozyme may help differentiate Crohn's disease from idiopathic ulcerative colitis.

1150 ASSESSMENT OF AMOEBIASIS: SERODIAGNOSIS BY IMMUNOFLUORESCENCE WITH LYOPHILIZED ENTAMOEBAE. *(E.)* Cox, J. C.; Nairn, R. C. (Monash Univ. Med. Sch., Melbourne, Australia). *J. Clin. Pathol.* 27(12):1018-1019, 1974.

Human sera were collected from cases of amebic colitis or amebic hepatitis. Slides of sera smears were prepared and the sandwich immunofluorescence method was applied using 0.1% Evans blue as counterstain to reduce background fluorescent staining and to give a distinct titration endpoint. FITC-labeled goat anti-human globulin was used as a conjugate at 5 mg/ml. The results obtained with the immunofluorescence antibody titration were reproducible. The four positive sera tested in 2 assessments gave the same immunofluorescence titer in 3 cases while the 4th varied by only 1 doubling dilution. Assessment of amebiasis was also made by using seramoeba latex agglutination kits as directed. Results when using the latex particle agglutination showed insufficient sensitivity in that 5 false negatives and 1 false positive were recorded. Thus, although the latex agglutination is a simpler procedure for serological diagnosis, it is less reliable and lacks sufficient sensitivity for routine use. This lack of sensitivity may be due to the fact that agglutination techniques are especially sensitive to IgM which has not been regularly detected in amebiasis serology. IgG is the major immunoglobulin found.

1151 COMPARATIVE STUDY BETWEEN ENDOSCOPY AND RADIOLOGY IN ACUTE UPPER GASTROINTESTINAL HAEMORRHAGE. *(E.)* Hoare, A. M. (King's Coll. Hosp., London, England). *Br. Med. J.* 1(5948):27-30, 1975.

1152 EMERGENCY GASTROINTESTINAL ANGIOGRAPHY: DIAGNOSIS AND TREATMENT. *(Fr.)* Jacot, C.; Girard, J. D.; Humair, L. (La Chaux-de-Fonds Hosp., Switzerland). *Helv. Med. Acta* 37(5/6):404, 1974.

1153 ROLE OF ENDOSCOPY IN DIAGNOSIS AND THERAPY OF GASTROINTESTINAL PRECANCER. *(Ger.)* Ottenjann, R. (Neuperlach Munic. Hosp., Munich, Germany). *Langenbecks Arch. Chir.* 337:767-770, 1974.

1154 RADIOIMMUNOASSAY OF ALPHA-FETOPROTEIN. V. INFLUENCE OF AGE ON THE PHYSIOLOGICAL SERUM ALPHA-FETOPROTEIN LEVEL IN MAN AND IN RAT. *(E.)* Masseyeff, R.; Gilli, J.; Krebs, B.; Bonet, C.; Zrihen, H. (Fac. Med., Nice, France). *Biomedicine* 21(8):353-357, 1974.

1155 TECHNIQUE, INTERPRETATIVE PRINCIPLES, COMPLICATIONS, AND INDICATIONS FOR SELECTIVE ABDOMINAL ARTERIOGRAPHY. *(Sp.)* Garcia Diaz, S.; Cantillana Martinez, J. (Fac. Med., Seville, Spain). *Rev. Esp. Enferm. Apar. Dig.* 44(3):207-218, 1974.

1156 ENDOSCOPY IN PATIENTS WITH UPPER GASTROINTESTINAL BLEEDING. EXPERIENCE WITH 386 PATIENTS. *(Fr.)* Bodin, F.; Lichtenstein, H.; Roudier, M.; Conte, M. (St. Antoine Hosp., Paris, France). *Arch. Fr. Mal. App. Dig.* 63(3):385-389, 1974.

1157 FIBEROPTIC GASTROINTESTINAL ENDOSCOPY IN INFANTS AND CHILDREN. *(E.)* Gleason, Jr., W. A.; Tedesco, F. J.; Keating, J. P.; Goldstein, P. D. (Washington Univ. Sch. Med., St. Louis, Mo.). *J. Pediatr.* 85(6):810-813, 1974.

1158 RESULTS OF COMBINED CYTOLOGIC AND HISTOLOGIC EXAMINATIONS IN GUIDED BIOPSIES OF 438 TUMORS OF THE STOMACH AND ESOPHAGUS. *(Ger.)* Ostertag, H.; Atay, Z.; Georgii, A. (Med. Coll., Hannover, Germany). *Verh. Dtsch. Ges. Pathol.* 57:299-301, 1973.

1159 EXFOLIATIVE CYTOLOGY OF THE GASTROINTESTINAL TRACT (REPORT). *(Ger.)* Witte, S. (Deaconess Hosp., Karlsruhe-Rüppruhr, Germany). *Verh. Dtsch. Ges. Pathol.* 57:126-138, 1973.

1160 THE COMBINED USE OF CYTOLOGY AND HISTOMORPHOLOGY IN THE DIAGNOSIS OF STOMACH DISEASE. *(Ger.)* Pantucek, F. (Pathol. Inst., Univ. Vienna, Austria). *Verh. Dtsch. Ges. Pathol.* 57: 301-305, 1973.

1161 TECHNIQUE OF AND PROSPECTS FOR GASTROSCOPIC SNARE BIOPSIES. *(Ger.)* Lindner, H.; Mitschke, H. (DRK Hosp., Hamburg, Germany). *Dtsch. Med. Wochenschr.* 99(10):468-469, 1974.

1162 ENDOSCOPIC SNARE BIOPSIES OF THE STOMACH. *(Ger.)* Deyhle, P. (Dep. Intern. Med., Univ. Zurich, Switzerland). *Dtsch. Med. Wochenschr.* 99(10):464-465, 1974.

1163 MASSIVE HEMORRHAGES FROM GASTRIC AND DUODENAL ULCERS: EMERGENCY DIAGNOSIS. *(Ger.)* Eichfuss, H. P.; Soehendra, N.; Wehling, H. (Surg. Clin., Univ. Hamburg, Germany). *Dtsch. Med. Wochenschr.* 99(35):1729-1731, 1974.

1164 CELLULAR CHANGES IN GASTRIC ULCERS AS SEEN BY EXFOLIATIVE CYTOLOGY. *(Ger.)* Schneider, M. L.; Maass, E. G.; Wiendl, H. J. (Inst. Clin. Surg., Tech. Coll., Munich, Germany). *Verh. Dtsch. Ges. Pathol.* 57:297-298, 1973.

1165 RADIOLOGIC EXAMINATION OF THE SMALL INTESTINE. *(Ger.)* Lenz, H. (St. Antonius Hosp., Eschweiler, Germany). *Radiologe* 14(9):393-403, 1974.

1166 PROBLEMS IN THE RADIOLOGICAL DIAGNOSIS OF TUMORS OF THE SMALL INTESTINE. *(Fr.)* Queloz, J.; Realini, S.; Candardjis, G. (Cantonal Hosp., Lausanne, Switzerland). *Arch. Fr. Mal. App. Dig.* 63(4):269-278, 1974.

1167 ENDOSCOPY OF THE SMALL INTESTINE. *(Ger.)* Fruhmorgen, P.; Classen, M. (Med. Clin., Univ. Erlangen-Nuremberg, Erlangen, Germany). *Radiologe* 14(9):404-408, 1974.

1168 THE IMPORTANCE OF CERTAIN ANOMALIES OF THE DUODENUM AND BILIO-PANCREATIC QUADRANGLE IN ENDOSCOPIC EXPLORATION OF THE DUODENAL PAPILLA. *(It.)* Farello, G. A.; Fratton, A.; Querci, G.; Della Rovere, G.; Gandolfi, P. (Inst. Surg. Pathol., Univ. Padua, Verona, Italy). *Chir. Gastroenterol.* 8(1):35-53, 1974.

1169 TECHNIQUE FOR BIOPSIES OF THE SMALL INTESTINE MUCOSA: PREMEDICATION AND OPERATION OF THE CAPSULE. *(Ger.)* Gaze, H. (no affil.). *Helv. Paediatr. Acta* (suppl) 33:23-24, 1974.

1170 COMPUTER-AIDED DIAGNOSIS OF LOWER GASTROINTESTINAL TRACT DISORDERS. *(E.)* de Dombal, F. T.; Clamp, S. E.; Leaper, D. J.; Staniland, J. R.; Horrocks, J. C. (Univ. Dep. Surg., Leeds, England). *Gastroenterology* 68(2):252-260, 1975.

1171 MESENTERIC ARTERIOGRAPHY IN INTESTINAL DISEASE. *(Sp.)* Garcia Diaz, S.; Cantillana Martinez, J. (Fac. Med., Univ. Seville, Spain). *Rev. Esp. Enferm. Apar. Dig.* 44(4):243-250, 1974.

1172 ENDOSCOPIC PANCREATOGRAPHY. ITS VALUE IN PREOPERATIVE AND POSTOPERATIVE ASSESSMENT OF PANCREATIC DISEASE. *(E.)* Cooperman, A. M.; Sivak, M. V.; Sullivan, Jr., B. H.; Hermann, R. E. (Cleveland Clin., Ohio). *Am. J. Surg.* 129(1):38-43, 1975.

1173 USING SCINTIGRAPHY IN PANCREATIC DIAGNOSIS. *(Ger.)* Bekier, A.; Berkovits, G. (Cantonal Hosp., St. Gallen, Switzerland). *Schweiz. Med. Wochenschr.* 104(51):1914-1917, 1974.

1174 THE DIAGNOSTIC SIGNIFICANCE OF EXCRETORY PANCREATIC TESTS. *(E.)* Bondar, Z. A.; Tuzhilin, S. A. (1st Moscow Med. Inst., USSR). *Am. J. Gastroenterol.* 62(6):488-496, 1974.

1175 CYTOLOGICAL DIAGNOSIS OF LOCALIZED TUMORS IN THE PANCREAS BY MEANS OF FINE NEEDLE BIOPSY DURING SURGERY. *(Ger.)* Bodner, E.; Lederer, B. (Surg. Clin., Univ. Innsbruck, Austria). *Verh. Dtsch. Ges. Pathol.* 57:377-379, 1973.

1176 CYTOLOGY OF THE PANCREAS AND PANCREATIC NEOPLASMS BY FINE NEEDLE ASPIRATION BIOPSY DURING PERITONEOSCOPY. *(Ger.)* Kirstaedter, H. J.; Meyer-Burg, J. (Steglitz Clin., Free Univ. Berlin, Germany). *Verh. Dtsch. Ges. Pathol.* 57:379-383, 1973.

1177 DOUBLE CONTRAST ELECTRORÖENTGENOGRAPHY (XERORADIOGRAPHY) OF THE LARGE INTESTINE. *(Ger.)* Kuncevic, D. E. (Lith. Clin. Radiol., Vilnius, USSR). *Radiol. Diagn. (Berl.)* 14(5):613-618, 1973.

1178 DOES A COMBINATION OF PANCREATIC SCINTIGRAPHY AND HYPOTONIC DUODENOGRAPHY IMPROVE DIAGNOSTIC INFORMATION? *(Ger.)* Luschnitz, E.; Taubert, W.; Lange, H. (Dep. Med., Karl Marx Univ., Leipzig, Germany). *Radiol. Diagn. (Berl.)* 14(6):757, 1973.

1179 SCINTILLATION SCANNING IN THE DIAGNOSIS OF POLYCYSTIC LIVER AND ITS SURGICAL TREATMENT. *(Rus.)* Marenko, T. P.; Kasatkin, Iu. N.; Esebaev, S. R. (Cent. Inst. Postgrad. Med., Moscow, USSR). *Vestn. Khir.* 113(10):39-44, 1974.

1180 LIVER SCANNING WITH ^{99m}Tc-Sn COLLOID. *(Jpn.)* Yamagishi, Y.; Honda, K.; Watanabe, H.; Shiiba, S.; Yukutake, J.; Karasawa, M. (Nippon Med. Sch., Tokyo, Japan). *Radioisotopes* 23(10):582-584, 1974.

1181 HEPATIC SCINTIGRAPHY AND THE LIVER BLOOD FLOW INDEX (K_L) WITH ^{99m}Tc COMPOUNDS (^{99m}Tc-Sn-PHYTATE AND ^{99m}Tc-Sn COLLOID) FOR DIAGNOSTIC EVALUATION OF DIFFUSE LIVER DISEASE: A COMPARISON OF ^{198}Au COLLOID AND ^{99m}Tc COMPOUNDS. *(Jpn.)* Miyamae, T. (Saitama Med. Sch., Moroyama, Japan). *Radioisotopes* 23(10):570-575, 1974.

1182 HEPATIC SCINTIGRAPHY USING ^{99m}Tc-Sn-COLLOID PREPARED ELECTROLYTICALLY. *(Jpn.)* Kaneko, M.; Tomita, T.; Yamamoto, C.; Sasaki, T.; Watanabe, M.; Mishima, A.; Tamiya, T.; Kato, S.; Yamaguchi, H. (Nagoya Univ. Hosp., Japan). *Radioisotopes* 23(10):576-581, 1974.

1183 HEPATIC AND CEREBRAL SCINTIGRAPHY WITH THE ^{113m}In KIT. *(Jpn.)* Yoshii, H.; Tateshi, K.; Fukui, K.; Hirota, Y.; Matsumoto, M.; Katayama, K. (Kumamoto Univ. Med. Sch., Japan). *Radioisotopes* 23(10):585-587, 1974.

1184 NONSPECIFICITY OF THE RADIOCOLLOID HEPATIC "HOTSPOT" FOR SUPERIOR VENA CAVAL OBSTRUCTION. *(E.)* Hattner, R. S.; Shames, D. M. (Univ. California Med. Cent., San Francisco). *J. Nucl. Med.* 15(11):1041-1043, 1974.

1185 THE VALUE OF ARTERIOGRAPHY IN SURGICAL DISEASES OF THE LIVER AND SPLEEN. *(Fr.)* Essinger, A.; Fluckiger, A. (Inst. Med. Radiol., Univ. Lausanne, Switzerland). *Helv. Chir. Acta* 41(5/6): 570-586, 1974.

1186 EFFECT OF PORTAL INJECTION OF LIPIODOL ULTRAFLUID ON CELL STRUCTURE IN THE LIVER. *(Fr.)* Garcia Diaz, S.; Arevalo, E.; Pera Madrazo, C. (Surg. Clin., Univ. Seville, Spain). *Lyon Chir.* 70(5):356-358, 1974.

1187 HEPATOGRAPHY USING INTRAARTERIAL INJECTION OF IODINE-CONTAINING OIL EMULSIONS. *(Fr.)* Lamarque, J. L.; Senac, J. P.; Bruel, J. M.; de Sars, P.; Boulet, P. (St. Eloi Clin., Montpellier, France). *Nouv. Presse Med.* 4(4):263-267, 1975.

1188 LIVER BIOPSIES IN THE FRAMEWORK OF ABDOMINAL SURGERY: VIEWPOINT OF THE PATHOLOGIST. *(Fr.)* Gardiol, D. (Inst. Pathol. Anat., Univ. Lausanne, Switzerland). *Helv. Chir. Acta* 41(5/6):595-599, 1974.

1189 FINE-NEEDLE ASPIRATION BIOPSY OF THE LIVER. *(Ger.)* Dannmeier, H. (Friedrich Ebert Hosp., Neumunster, Germany). *Verh. Dtsch. Ges. Pathol.* 57:243-246, 1973.

1190 A REVIEW OF OVER ONE THOUSAND CASES IN WHICH LAPAROSCOPY WAS CARRIED OUT FOR DIAGNOSIS OF DISEASE OF THE LIVER AND BILE DUCTS. *(It.)* Infranzi, A.; Sessa, R. (A. Cardarelli Gen. Reg. Hosp., Naples, Italy). *Chir. Gastroenterol.* 8(1):54-57, 1974.

1191 METABOLIC SPYING: LIVER AND BILIARY FUNCTION TEST WITH BENGAL ROSE. *(Ger.)* Subowski, G.; Rjasanskaja, G. (no affil.). *Exakt* (9):26-28, 1974.

1192 DETERMINATION OF LACTATE DEHYDROGENASE FRACTIONS IN THE DIAGNOSIS OF MALIGNANT METASTASES IN THE LIVER. *(Pol.)* Alwasiak, Z.; Dziewaltowska, D.; Joss, A. (Acad. Med., Lodz, Poland). *Wiad. Lek.* 27(14):1265-1268, 1974.

1193 SERUM IMMUNOGLOBULIN CONCENTRATIONS IN RELATION TO THE INTRAHEPATIC MESENCHYMAL CELL REACTION. *(Ger.)* Nilius, R.; Rath, F. W.; Zipprich, B. (1st Med. Clin., Martin Luther Univ., Halle-Wittenberg, Germany). *Dtsch. Z. Verdau. Stoffwechselkr.* 34(1):37-42, 1974.

1194 SEROLOGICAL DIAGNOSIS OF AMEBIASIS BY A RAPID AGGLUTINATION TEST USING SENSITIZED LATEX PARTICLES: RESULTS OF 462 EXAMINATIONS AND A COMPARISON WITH THE INDIRECT IMMUNOFLUORESCENCE REACTION. *(Fr.)* Ambroise-Thomas, P. (Hosp. Cent., Univ. Grenoble, France). *Bull. Soc. Pathol. Exot.* 67(2): 156-166, 1974.

1195 THE EPIDEMIOLOGY OF CHRONIC LIVER DISEASE AS REFLECTED BY A COMBINATION OF BIOCHEMICAL SCREENING EXAMINATIONS. *(Ger.)* Ehrke, D.; Bohnensack, G.; Depersdorff, J.; Friedemann, H.; Linneke, P.; Schmudlach, H. O.; Storm, H.; Thiele, H. J. (Schwerin Reg. Hosp., Germany). *Z. Gesamte Inn. Med.* 29(23): 969-972, 1974.

1196 LAPAROSCOPIC DIAGNOSIS OF HEPATITIS. *(Sp.)* Solis Herruzo, J. A. (1st October Munic. Sanit., Madrid, Spain). *Rev. Esp. Enferm. Apar. Dig.* 44(4):351-364, 1974.

1197 WEDGED HEPATIC VENOGRAPHY. A COMPARATIVE STUDY OF CIRRHOSIS AND HEPATIC SCHISTOSOMIASIS. *(E.)* Guelrud, M.; Beker, S. (Univ. Hosp. Caracas, Cent. Univ. Venezuela). *Am. J. Gastroenterol.* 62(6):504-508, 1974.

1198 ARTERIAL PORTOGRAPHY FOR PORTAL HYPERTENSION. *(Ger.)* Beranek, I.; Belan, A.; Vosmik, J. (Inst. Clin. Exp. Med., Prague, Czechoslovakia). *Fortschr. Geb. Roentgenstr. Nuklearmed.* 120(6):673-680, 1974.

1199 INDIRECT (ARTERIAL) SPLENO-PORTOGRAPHY AND PORTOGRAPHY IN THE DIAGNOSIS OF PREHEPATIC BLOCKS. *(Ger.)* Frommhold, H. (Radiol. Clin., Univ. Bonn, Germany). *Fortschr. Geb. Roentgenstr. Nuklearmed.* 120(6):662-672, 1974.

1200 CLINICAL EVALUATION OF INFUSION CHOLANGIOGRAPHY. *(Pol.)* Szulc, E. J. (T. Chalubinski Hosp., Czestochowa, Poland). *Wiad. Lek.* 27(19): 1707-1712, 1974.

1201 PERCUTANEOUS TRANSHEPATIC CHOLANGIOGRAPHY. *(E.)* Hung, V. (Bach-mai Hosp., Hanoi, Vietnam). *Radiol. Diagn. (Berl.)* 14(4):471-476, 1973.

1202 PERCUTANEOUS TRANSHEPATIC CHOLANGIOGRAPHY. *(E.)* Cline, R. L.; Matallana, R. H.; Crummy, A. B. (Univ. Wisconsin Cent. Health Sci., Madison). *Australas. Radiol.* 18(2):191-197, 1974.

1203 ENDOSCOPIC VISUALIZATION OF THE PANCREATIC AND BILIARY DUCTS. *(E.)* Waye, J. D. (Mt. Sinai Hosp., City Univ. New York, N.Y.). *Mt. Sinai J. Med. NY* 42(1):35-56, 1975.

1204 ENDOSCOPIC RETROGRADE CHOLANGIOPANCREATOGRAPHY IN DIAGNOSIS OF BILIARY DISEASE. *(Ger.)* Deyhle, P.; Nette, L.; Ammann, R. (Dep. Intern. Med., Univ. Zurich, Switzerland). *Helv. Chir. Acta* 41(5/6):587-593, 1974.

1205 CHOLANGIOSCOPY DURING SURGERY. *(Ger.)* Berchtold, R.; Ghielmetti, C. (Inselspital, Bern, Switzerland). *Helv. Chir. Acta* 41(5/6):601-602, 1974.

1206 INDICATIONS FOR PERCUTANEOUS TRANSHEPATIC CHOLANGIOGRAPHY (PTC) AND ENDOSCOPIC RETROGRADE PANCREATICOCHOLANGIOGRAPHY (ERPC). *(Ger.)* Weber, A. P.; Schwarz, H. (Limmattal Hosp., Schlieren, Switzerland). *Helv. Chir. Acta* 41(5/6):619-623, 1974.

1207 SIMPLIFIED CHOLANGIOMANOMETRY AND FLOW MEASUREMENT DURING SURGERY. *(Ger.)* Tondelli, P.; Allgower, M. (Cantonal Hosp., Basel, Switzerland). *Helv. Chir. Acta* 41(5/6):609-613, 1974.

1208 CHOLANGIOGRAPHY AND RADIOMANOMETRY DURING SURGERY. *(Fr.)* Rohner, A. (Surg. Clin., Univ. Geneva, Switzerland). *Helv. Chir. Acta* 41 (5/6):603-607, 1974.

1209 ULTRASONIC EXPLORATION OF DILATATION OF THE COMMON BILE DUCT. ITS PLACE IN THE RADIOLOGY OF JAUNDICE: 21 CASES. *(Fr.)* Weill, F.; Bourgoin, A.; Aucant, D.; Eisencher, A.; Faivre, M.; Gillet, M. (Univ. Hosp. Cent., Besancon, France). *Arch. Fr. Mal. App. Dig.* 63(6):453-472, 1975.

1210 DIAGNOSTIC SIGNIFICANCE OF INTRAVENOUS AND INFUSION CHOLANGIOGRAPHY IN BILIARY DISEASES. *(Pol.)* Rzymski, K.; Zwozdziak, L. (Acad. Med., Poznan, Poland). *Wiad. Lek.* 27(7):609-614, 1974.

See also, 1211, 1239, 1251, 1341, 1368, 1418, 1421, 1472, 1483, 1484, 1493, 1524, 1570.

GASTROINTESTINAL DISEASES

ESOPHAGUS

1211 ESOPHAGOSCOPY AS A SOURCE OF PSEUDOMONAS AERUGINOSA SEPSIS IN PATIENTS WITH ACUTE LEUKEMIA: THE NEED FOR STERILIZATION OF ENDOSCOPES. *(E.)* Greene, W. H.; Moody, M.; Hartley, R.; Effman, E.; Aisner, J.; Young, V. M.; Wiernik, P. H. (Natl. Cancer Inst., Baltimore, Md.). *Gastroenterology* 67(5):912-919, 1974.

Culture reports and clinical records of the 12 patients who had undergone endoscopy with a flexible fiberoptic esophagoscope since its purchase in July, 1972 were reviewed to determine if this procedure could be a source of hospital-acquired infection. This review was occasioned by the appearance of *Pseudomonas aeruginosa* sepsis shortly after esophagoscopy in 1 of the patients. Among the 12 patients reviewed, only 3, all with acute leukemia, actually had cultures of esophageal washings (4 times) or tissue (2 times). In each instance, *P. aeruginosa*, of either serotype 5 or 7, was recovered, representing, on 5 of 6 occasions, either the first isolate of any *P. aeruginosa* or the first isolate of that particular serotype recovered from the patient after a minimum of 1 month of culturing. Esophageal mucosal biopsy was performed at the time of endoscopy in 2 of 3 leukemia patients. Each was severely granulocytopenic and in both patients fatal pseudomonas septicemia followed endoscopy, 12 hr and 3 days later. The absence of septicemia in the third patient may be related to a lesser degree of granulocytopenia, the absence of esophageal pathology, and/or to the failure to penetrate the mucosa for biopsy. Cultures of the esophagoscope and of the endoscopy room revealed widespread contamination by enteric organisms, including *P. aeruginosa*; routine handling of the instrument did not correspond to aseptic techniques. It is concluded that endoscopy, particularly with biopsy, in the compromised host carries the dual risk of inducing sepsis from endogenous flora and of introducing invasive nosocomial organisms, especially *P. aeruginosa*. It is recommended that among endoscopy subjects with a significant degree of compromise of host defenses, especially granulocytopenia, sterile precautions, including sterile water, bowls, sterile draping of surfaces, and effective decontamination of the instrument, be employed.

1212 INTRALUMINAL ESOPHAGEAL REWARMING IN NORMALS AND IN PATIENTS WITH CONNECTIVE TISSUE DISEASE. *(E.)* Harell, G. S.; Gold, J. A.; Fries, J. F.; Bradley, B.; Zboralske, F. F. (Stanford Univ. Sch. Med., Calif.). *Invest. Radiol.* 9(3):186-192, 1974.

To determine esophageal involvement in patients with connective tissue disorders, intraluminal esophageal rewarming curves were obtained after ingestion of a bolus of 0.5 C water while the subjects were seated upright. Thermistors were inserted through the nose into the proximal and distal esophagus of 15 normal controls, 4 patients with scleroderma, 3 with systemic lupus erythematosus, 1 with mixed connective tissue disorder, and 2 with Raynaud's disease. The measurement time, T 1/2, required for a thermistor to record a temperature half-way between the minimum temperature reached after ice water ingestion and the baseline temperature was determined for each swallow. Semilogarithmic intraluminal rewarming graphs from both groups had 1 or more straight line segments, indicating that the resistors rewarmed exponentially. There was a significant prolongation of the distal T 1/2 when the patient group (as a whole) was compared with the controls. The results indicate that the ice water remains in the distal esophagus of the patient longer because of abnormal esophageal motility. There was no pooling in the proximal esophagus. Following propantheline bromide injection in 2 controls, the distal 2/3 of the esophagus was moderately dilated, amotile, and had significant fluid retention. Pooling (and subsequent increased T 1/2) in the patient group may occur due to loss of peristalsis in the distal esophagus. Esophageal dilatation alone is probably not a cause of prolonged distal T 1/2. An alteration in esophageal blood flow is associated with the smooth muscle atrophy and motility disorder found in systemic sclerosis. These studies suggest that intraluminal rewarming curves are an indication of esophageal involvement in connective tissue disorders. The data do not support or refute the hypothesis that motility disorders are due to altered esophageal blood flow.

1213 UNRESECTABLE CARCINOMA OF THE OESOPHAGUS. *(E.)* Ong, G. B. (Dep. Surg., Univ. Hong Kong). *Ann. R. Coll. Surg. Engl.* 56(1):3-14, 1975.

1214 MALLORY-WEISS SYNDROME. *(E.)* Wychulis, A. R.; Sasso, A. (St. Michael's Med. Cent., Newark, N.J.). *Arch. Surg.* 107(6):868-871, 1973.

See also, 1125, 1156, 1157, 1158, 1159, 1534, 1545.

STOMACH

1215 EFFECT OF JEJUNAL INTERPOSITION ON ALCOHOL ABSORPTION FOLLOWING SUBTOTAL GASTRECTOMY. *(E.)* Rahbar, A.; Mendoza, Jr., C. B.; Zimmermann, B. (West Virginia Univ. Sch. Med., Morgantown). *Am. Surg.* 40(12):702-703, 1974.

The effect of jejunal interposition on absorption of alcohol was studied in gastrectomized dogs. Four groups of dogs were used: group 1 served as controls without operation; group 2, 75% gastrectomy with Billroth II reconstruction; group 3, same as group 2 plus interposition of a segment of jejunum about 25 cm in length between the gastric remnant and the duodenum; group 4, isolated jejunal pouch 25 cm in length. Each animal received 1 ml of 25% ethanol/lb. This was delivered *via* stomach tube in groups 1, 2, and 3, and *via* the stoma of the jejunal pouch in group 4. Blood alcohol levels in normal and gastrectomized dogs were not significantly different. Dogs with the interposed jejunal segment showed a statistically significant decrease in blood alcohol levels compared with normal and gastrectomized dogs of 50, 66, and 65 mg/100 ml after 30 min, resp. Dogs with the isolated jejunum showed even greater decreases in blood alcohol levels (36 mg/100 ml at 30 min) with a slowly rising curve. Thus it is concluded that the interposition of 25 cm of jejunum between the gastric remnant and duodenum decreases the blood alcohol levels after ingestion. However, it is felt that patients who have undergone gastric operation should still be warned of a possible change in tolerance to alcohol consumption.

1216 ISOLATED GASTRIC PNEUMATOSIS. *(E.)* Holgersen, L. O.; Borns, P. F.; Srouji, M. N. (Child. Hosp. Philadelphia, Pa.). *J. Pediatr. Surg.* 9(6):813-816, 1974.

Four recent cases of isolated gastric pneumatosis (3 males and 1 female, aged 7 days to 11 weeks) are presented. Three patients had pyloric stenosis with hematemesis present in 2, and coffee-ground vomitus suggestive of blood in the third. The 4th patient, a 2400 g boy delivered of a pre-eclamptic primapara mother, experienced laryngospasm, apnea, poor tone, and depressed reflexes requiring resuscitation. Early feeding caused distention, particularly in the left upper quadrant. A nasogastric tube brought up a large amount of air and brownish material mixed with blood. Plain films of the abdomen at 4 days of age showed a dilated stomach with gastric-wall air, and diminished air in the remainder of the gastrointestinal tract. Upper gastrointestinal series showed no passage of barium from the stomach after 6 hr. At operation the appearance of the gastric wall and pylorus were normal and a gastrostomy was performed. The time between symptoms and the radiographic documentation of air in the gastric wall of these patients varied from 3 to 30 days. The distribution of air within the stomach wall was relatively symmetrical, but the fundus contained a greater amount of air in 3 of the patients. One patient had the additional finding of free intraperitoneal air, although no signs of peritonitis were present. The gastric pneumatosis disappeared radiographically 2-7 days after gastric decompression. None of these 4 patients was seriously ill, and all had a smooth clinical course with survival. They had in common the presence of increased intragastric pressure and gastric outlet obstruction, 3 mechanical and 1 functional. The increased intragastric pressure was involved in the mechanism of the formation of air in the gastric wall, and a mucosal tear with submucosal dissection of the air might explain the association of gastric pneumatosis with increased intragastric pressure. This view is supported by the prompt resolution of the pneumatosis following gastric decompression.

1217 MALLORY WEISS SYNDROME--DIAGNOSIS AND TREATMENT. *(E.)* Jensen, S. L.; Pagel, J.; Nielsen, O. V. (Rigshosp., Univ. Copenhagen, Denmark). *Scand. J. Gastroenterol.* 9(29):14, 1974.

See also, 1135, 1156, 1157, 1158, 1160, 1161, 1162, 1317, 1452, 1534, 1536, 1545, 1546.

STOMACH

Peptic Ulcers

1218 ORAL METIAMIDE AS AN EFFECTIVE INHIBITOR OF GASTRIC ACID SECRETION IN MAN. *(E.)* Barbezat, G. O.; Bank, S.; Clain, J.; Novis, B.; Marks, I. N. (Groote Schuur Hosp., Cape Town, South Africa). *S. Afr. Med. J.* 48(48):2018-2021, 1974.

The effect or oral metiamide on gastric acid secretion stimulated by maximal doses of pentagastrin (6 µg/kg/hr) was studied in 9 duodenal ulcer patients (aged 20-53 yr). Introduction of metiamide tablets (200 mg, crushed, suspended in 100 ml 0.03 M HCl solution) into the stomach *via* a nasogastric tube, caused significant reductions in basal and pentagastrin stimulated volume output and acid concentration. Maximal acid output and peak acid output were reduced by 51.8% and 51% resp., a highly significant inhibition of gastric acid secretion. This represents the effect of the drug 2-3 hr after entering the stomach *via* p.o. administration. Metiamide is well absorbed from the gastrointestinal tract and there were no side-effects noted with the 200 mg dose level. These results indicate that metiamide is highly effective in reducing pentagastrin stimulated gastric acid secretion when taken orally.

1219 A COMPARISON OF GASTRIC EMPTYING AT 3 AND 12 MONTHS AFTER PROXIMAL GASTRIC OR SELECTIVE VAGOTOMY WITHOUT PYLOROPLASTY. *(E.)* Donovan, I. A.; Clarke, R. J.; Gunn, I. F.; Alexander-Williams, J. (Gen. Hosp., Birmingham, England). *Br. J. Surg.* 61(11):889-892, 1974.

A prospective study was performed to determine whether the early postoperative disturbance of gastric emptying after vagotomy changed with the passage of time. In 20 patients the rate of emptying of 750 ml of a 10% dextrose meal was measured by a double sampling dye dilution technique before and 3 months after proximal gastric vagotomy or selective vagotomy without a drainage procedure. Ten of these patients agreed to a further emptying study at 12 months after operation. Profound changes in the pattern of emptying occurred as a result of either operation. The first measurement of gastric volume, made 10 min after the meal, showed that gastric incontinence is no more pronounced after selective vagotomy than after proximal gastric vagotomy. In neither group of patients did the volume emptied at 10 min change between 3 and 12 months after operation, suggesting that it is unlikely that any improvement in the gastric incontinence will occur. After this initial phase of incontinence, the emptying of the remainder of the meal, probably a result of antral muscular action, showed no significant change at 3 or 12 months in patients receiving proximal gastric vagotomy. However, selective vagotomy patients, in whom the antral musculature is denervated, showed an impairment of the ability of the stomach to empty actively as reflected in the prolonged total emptying time. The fact that at 12 months after operation this increase in total emptying time had lessened in some patients suggests that the muscular action of the denervated antrum may recover to some extent with time.

1220 GASTRIN RESPONSE TO FOOD IN DUODENAL ULCER PATIENTS BEFORE AND AFTER SELECTIVE OR HIGHLY SELECTIVE VAGOTOMY. *(E.)* Stadil, F.; Rehfeld, J. F.; Christiansen, P. M.; Kronborg, O. (Rigshosp., Copenhagen, Denmark). *Br. J. Surg.* 61(11):884-888, 1974.

The role of the vagus in the gastrin response to a steak meal was investigated in 61 duodenal ulcer patients. Thirty patients (23 males and 7 females, mean age 46 yr) were studied before and 2 weeks after selective vagotomy with a Weinberg pyloroplasty. Basal and stimulated gastrin concentrations in serum, as determined by radioimmunoassay, were higher postoperatively (87 pre-operatively/111 postoperatively for basal and 243/341 for peak, pg/ml). Another group of 30 patients (21 males and 9 females, mean age 48 yr) were studied before and after highly selective vagotomy without drainage. Basal and stimulated gastrin concentrations were also higher postoperatively in these patients (94/133 and 277/343 pg/ml, resp.). The differences between the two groups of patients were insignificant. Between individual cases, gastrin responses varied markedly, but pre- and postoperative responses were positively related within the same patient. No differences were noted between patients with positive or negative Hollander responses to insulin. In 6 patients, 1 after highly selective vagotomy and 5 after selective vagotomy, studied 2 and 14 weeks postoperatively, no difference was apparent. Thus it is concluded that antral branches of the vagi play only a minor role, if any, in the gastrin response to food.

1221 NEUROLOGICAL SYMPTOMS AND SIGNS IN A SELECTED GROUP OF PARTIALLY GASTRECTOMIZED PATIENTS WITH PARTICULAR REFERENCE TO B_{12} DEFICIENCY. *(E.)* Roos, D. (Dep. Neurol., Univ. Copenhagen, Denmark). *Acta Neurol. Scand.* 50(6):719-752, 1974.

The neurological manifestations observed in 42 partially gastrectomized patients (9 women and 33 men, mean age 56.5 yr) with serum B_{12} levels below 200 pg/ml were investigated. A Billroth I operation had been performed in 8 patients, a Billroth II in 30. In 4 cases the operation consisted of pyloroplasty plus total vagotomy. Of these 42 patients, 17 were anemic, including 6 with reduced serum iron, sometimes accompanied by a high serum transferrin. Only 6 showed the classical appearance of megaloblastic bone marrow. Relevant descriptions of sensory or motor symptoms after operation were given by 31 patients. Clinical neurological examination revealed abnormal neurological signs in 35 (83%), presumably due to B_{12} deficiency. Generally, the somato-neurological symptoms were modest, overshadowed by severe gastrointestinal symptoms or by emotional and intellectual impairment. Thirty patients complained of constant dyspeptic symptoms, paresthesia (24), hypoesthesia and dysesthesia (12), pain (3), dizziness (16), disturbances of coordination (7), and localized weakness (3). A surprisingly large number of patients having serum B_{12} below 200 pg/ml were impotent, 11 of the 33 men. In 1 patient this was combined with urinary incontinence. Serum B_{12} averaged 142 pg/ml in these 11 patients. The dyspeptic symptoms could be grouped into the afferent loop syndrome, the small stomach syndrome and dumping. Vomiting of bile did not occur. Pain related to meals was noted in 27 patients. Dumping was present in 22, 20 of whom also suffered from the small stomach syndrome. Twenty-three suffered from diarrhea, often imperious and often provoked by meals. In 15 of 23 patients the stools were loose and fatty. Thirty-three patients continued with injections of vitamin B_{12}. Twenty-six of them reported subjective improvement, especially regarding peripheral extremity symptoms. Eighteen also experienced objective improvement. Considering the serious and gradually irreversible mental and neurological defects that vitamin B_{12} deficiency can entail, vitamin B_{12} therapy of a partially gastrectomized patient with a low serum B_{12} level is the only correct solution.

1222 NEUROLOGICAL COMPLICATIONS IN AN UNSELECTED GROUP OF PATIENTS PARTIALLY GASTRECTOMIZED FOR GASTRIC ULCER. *(E.)* Roos, D. (Bispebjerg Hosp., Copenhagen, Denmark). *Acta Neurol. Scand.* 50(6): 753-773, 1974.

A clinical neurological follow-up study was conducted on 128 patients with a history of surgery for benign gastric ulcer an average of 12 yr previously. At operation 127 patients had a gastric ulcer situated in the corpus (92) or antrum (37) cases; 2 patients had ulcers in both sites. One patient had severe gastroduodenitis. Duodenal ulcer was also present in 23 patients. In 32 cases the primary surgery was a Billroth I and in 92 cases, a Billroth II. In 2 cases the Billroth I was later converted to a II. The

age range at operation was from 29 to over 50 yr. At follow-up there were 42 females and 86 males, aged 41-90 yr. Neurological symptoms present at follow-up included dizziness (39 patients), paraesthesiae in toes and fingers (54), nocturnal cramps in calves (3), piercing, deep, often nocturnal pain in upper or lower limbs (5), hypoesthesia, anesthesia, or dysesthesia (24), localized weakness (12), uncoordination (5), loss of potency (5), and visual impairment (5). Among these 128 patients, 44 had signs of myelopathy, peripheral neuropathy or severe intellectual impairment, presumably due to vitamin B_{12} deficiency. Severe intellectual impairment was found in 10 of 29 patients with serum B_{12} levels below 150 pg/ml. Four of these 44 patients were on vitamin B_{12} therapy. In the remaining 40 the serum vitamin B_{12} averaged 154 pg/ml. Out of the 44 affected patients 14 were in the serum B_{12} range 150-200 pg/ml. B_{12} therapy is recommended also for this group of "questionable reduction" in order to avoid irreversible cerebral and spinal lesions due to the B_{12} deficiency.

1223 HIGHLY SELECTIVE VAGOTOMY IN THE TREATMENT OF DUODENAL ULCER. *(Fr.)* Imperati, L.; Marinaccio, F.; Altieri, A. (Reg. Hosp., Foggia, Italy). *Nouv. Presse Med.* 24(29):1811-1813, 1974.

See also, 0860, 0872, 1156, 1157, 1163, 1164, 1174, 1440, 1451, 1471.

SMALL INTESTINE

1224 NEUROGENIC TUMORS OF THE SMALL INTESTINE. REVIEW OF THE LITERATURE AND REPORT OF A CASE WITH ENDOSCOPIC REMOVAL. *(E.)* Sivak, Jr., M. V.; Sullivan, Jr., B. H.; Farmer, R. G. (Cleveland Clin. Found., Ohio). *Gastroenterology* 68(2): 374-380, 1975.

Epigastric pain and melena prompted the study of a 62-yr-old woman with cutaneous neurofibromatosis, von Recklinghausen's disease. Roentgenographic and endoscopic examination demonstrated an ulcerated polypoid neurilemoma of the duodenum. It was removed by endoscopic electrosurgery. This is the first report of the endoscopic management of bleeding related to these tumors. Neurogenic small bowel tumors, while rare, must be considered in the differential diagnosis of gastrointestinal bleeding which has been localized to the small intestine by standard diagnostic measures or is of uncertain origin. Endoscopic polypectomy with electrocoagulation of an ulcerated tumor is proposed as a relatively safe measure for those lesions within the range of the instrument. If the procedure is not effective, laparotomy may be required.

1225 ANTIPERISTALTIC SEGMENTS FOR THE TREATMENT OF SHORT BOWEL SYNDROME. *(E.)* Pertsemlidis, D.; Kark, A. E. (Mt. Sinai Sch. Med., City Univ. New York, N.Y.). *Am. J. Gastroenterol.* 62(6):526-530, 1974.

The successful use of an antiperistaltic small bowel segment is demonstrated in a 33-yr-old woman with incapacitating fluid and electrolyte imbalance due to excessive jejunostomy losses. Two years prior to her current admission, she had been operated on for ileocolitis. The operation consisted of distal ileectomy (about 50 cm), subtotal colectomy and ileosigmoidostomy. The tentative pathological diagnosis was "ulcerative colitis". Complications required a second operation 14 months later at which time the ileosigmoidostomy was taken down, 85 cm of diseased ileum was resected, a jejunostomy was constructed and the lower sigmoid was brought out through a separate cutaneous exit (mucous fistula). Pathological diagnosis was changed to granulomatous ileocolitis (Crohn's disease). Radiographic examinations indicated that 120-150 cm of jejunum were left between the ligament of Treitz and the jejunal stoma. The intestinal transit time using barium meal was estimated to be 10 min, even though the remaining small bowel was free of disease. Uncontrollable water and electrolyte imbalance necessitated the creation of an antiperistaltic jejunal segment. A 14 cm segment was reversed, located about 12-15 cm from the jejunostomy, by the method of Rygick and Nasarov which permits the reversal of the segment without twisting the mesentery. On recovery she was able to maintain adequate fluid balance and to ingest solid food diet without excessive losses through jejunostomy. In the past 2 years she has remained well. Intermittent mild azotemia is corrected by increasing the oral fluid intake. It is suggested that the remaining 120-150 cm of normal jejunum would have been adequate for this patient had the colon been intact. The site for creating a reversed loop should be as distal in the small bowel as possible. However, the choice of the site for a reversed loop is dictated by biochemical rather than mechanical criteria.

1226 FAMILIAL MECONIUM ILEUS NOT ASSOCIATED WITH CYSTIC FIBROSIS. *(E.)* Dolan, Jr., T. F.; Touloukian, R. J. (Yale Univ. Sch. Med., New Haven, Conn.). *J. Pediatr. Surg.* 9(6):821-824, 1974.

The occurrence of meconium ileus is reported in 2 full-term male siblings. The mother had had an intestinal obstruction of unknown cause at 1 yr of age requiring surgery. In neither of these two cases was a characteristic meconium plug seen at operation or delivered by Gastrografin enema. Both children are now receiving regular diets and have normal growth and development without gastrointestinal or pulmonary problems. There is no suggestion of Hirschsprung's disease. The explanation for meconium obstruction in these patients is not known. Protein analysis of the meconium was not performed. It is felt that this may be the first report of familial meconium ileus without associated cystic

fibrosis of the pancreas or pancreatic insufficiency. These children supply proof that not all patients with meconium ileus will eventually prove to have cystic fibrosis. Definitive parental counseling should be delayed until an accurate sweat test can be performed, usually not possible until the infant is about 3-4 weeks of age.

1227 VASODILATORY DRUGS IN THE MANAGEMENT OF NONOCCLUSIVE BOWEL ISCHEMIA. *(E.)* Athanasoulis, C. A.; Wittenberg, J.; Bernstein, R.; Williams, L. F. (Boston City Hosp., Mass.). *Gastroenterology* 68(1):146-150, 1975.

A 75-yr-old female was hospitalized because of an acute myocardial infarction. An electrocardiogram showed marked digitalis toxicity with occasional premature ventricular contraction. On the 10th day, abdominal pain and rising temperature were apparent. An abdominal radiograph showed distention of the small and large bowel with thickening of the small bowel wall, suggesting bowel ischemia. After the diagnosis of nonocclusive mesenteric ischemia was firm, papaverine (0.4 mg/kg, or 30.0 mg) was rapidly injected as a bolus through the angiographic catheter into the superior mesenteric artery followed by papaverine infusion (0.8 mg/kg/hr) for 1 hr. Papaverine showed minimal dilation of jejunal arterial branches. Phenoxybenzamine (0.2 mg/kg) was injected as a bolus into the superior mesenteric artery followed by an infusion at a rate of 0.7 mg/kg/hr for 1 hr. Phenoxybenzamine showed reversal of the vasoconstriction with marked dilation of the superior mesenteric artery and all its branches. After 11 hr, phenoxybenzamine was again infused (0.7 mg/kg/hr). After 1 hr no effect on the size of the middle colic artery was noted. An emergency upper gastrointestinal series showed normal stomach and duodenum with distention of the proximal small bowel. At 18 hr after the initial infusion, the patient's condition was unchanged and the stool became guaiac-positive. At exploratory laparotomy a double barrel transverse colostomy was created. The patient recovered and, when released on the 46th hospital day, had no signs of nutritional difficulties. This approach of using resuscitative measures and delaying operative intervention in such cases, decreases the extent of small bowel resection and should also decrease the incidence of anastomotic leaks which complicate early resections. Operative intervention is still necessary as many of these patients do have full wall necrosis of a bowel segment and the nonoperative diagnosis of nonocclusive mesenteric ischemia remains one of presumption. The advantage in using papaverine is the lack of systemic effects at infusion rates of 1 mg/min for several hr or days. The disadvantage is that several hr are required before papaverine achieves its major effect. Thus it is concluded that a patient with the clinical, angiographic, operative, and pathological diagnosis of nonocclusive mesenteric ischemia can respond to direct superior mesenteric artery infusion of vasodilatory drugs.

1228 GASTROINTESTINAL HEMORRHAGE -- AN UNUSUAL MANIFESTATION OF NEUROFIBROMATOSIS. *(E.)* Devereux, R. B.; Koblenz, L. W.; Cipriano, P.; Gray, G. F. (New York Hosp., N.Y.). *Am. J. Med.* 58(1): 135-138, 1974.

A 29-yr-old Puerto Rican male presented with a 4 day history of melena, pregressive weakness and vomiting of bilious gastric contents. He had had multiple subcutaneous nodules and cafe au lait spots all his life. For 10 years prior to admission he experienced epigastric pain almost daily, which progressively worsened in the weeks before bleeding commenced. An x-ray series of the upper gastrointestinal tract and small bowel revealed only a small hiatus hernia. Superior mesenteric and inferior mesenteric arteriography disclosed a vascular tumor in the jejunum. Surgery demonstrated a single dumbbell-shaped intramural tumor which appeared to arise within the muscularis of the jejunum. About half the lesion protruded into the serosa whereas the other half compressed the lumen with attenuation of the mucosa in which there was a shallow erosion 3 mm in diameter. Microscopically the leiomyoma consisted of whorls of spindly cells with uniform nuclei without pleomorphism or mitotic figures. The appearance closely resembled that of intestinal smooth muscle. Epithelioid cell granulomas with giant cells, some containing fragments of *Schistosoma* eggs, were within the tumor. Similar granulomas were also in the intestinal mucosa. The patient remains well 1 yr after recovery, free of abdominal complaints. A small intestinal tumor must be highly suspect as the source of bleeding in any patient with neurofibromatosis, gastrointestinal hemorrhage, and a negative nasogastric aspirate. Arteriography should be the contrast procedure of first choice. Definitive resection should be undertaken in view of the frequency of recurrent life-threatening hemorrhage.

1229 AORTODUODENAL FISTULA: COMPLICATION OF RENAL ARTERY BYPASS GRAFT. *(E.)* Keeffe, E. B.; Krippaehne, W. W.; Rosch, J.; Melnyk, C. S. (Univ. Oregon Med. Sch., Portland). *Gastroenterology* 67(6):1240-1244, 1974.

A 35-yr-old female developed postpartum arterial hypertension. Angiography showed advanced medial fibromuscular hyperplasia of the right renal artery. This was treated with a Dacron bypass graft. The following year hypertension reappeared and graft occlusion was noted. The graft was shortened and repaired. Recurrent clotting in the graft 2 years later led to placement of a second bypass graft off the aorta. Hypertension returned and was managed medically. Three years later angiography demonstrated advanced medial hyperplasia of the right renal artery and occlusion of the second graft. The following year found medical management of her hypertension impossible, and surgical autotransplantation of her right kidney to the left iliac fossa was performed at which time the occluded second graft was ligated and left attached to the aorta as had been done with the first graft. At age 44 she was admitted for gastrointestinal bleeding. Over the next 2 months she was readmitted 3 additional times for gastrointestinal bleeding, and 10 days after her final admission she collapsed in shock from massive gastrointestinal bleeding and died during emergency

surgery. During this surgery an aortoduodenal fistula was discovered. This may be the first reported case of a fistula developing from the proximal anastomosis of renal bypass graft surgery. An aortointestinal fistula should be considered as a cause of gastrointestinal hemorrhage in patients with a history of renal artery bypass graft placement or any other arterial reconstructive surgery in proximity to the gastrointestinal tract. Prompt surgical exploration in the clinical setting of aneurysm, or former arterial or aortic vascular surgery, remains the best approach to diagnosis as well as therapy.

1230 PRIMARY CARCINOMA OF SMALL INTESTINE. *(Pol.)* Borowski, Z. (F. Krzystalowicz Dist. Hosp., Olkusz, Poland). *Wiad. Lek.* 27(11): 1037-1041, 1974.

1231 DUODENECTOMY IN CANCER OF DUODENUM AND THE MAJOR DUODENAL PAPILLA. *(Rus.)* Futoryan, E. S.; Shubin, B. M. (P. A. Gertsen Res. Inst. Oncol., Moscow, USSR). *Khirurgiia (Mosk.)* (1):69-72, 1974.

1232 DUODENAL LIPOMA. *(Fr.)* Suire, P.; Gousse, C. (no affil.). *J. Chir. (Paris)* 107(2): 177-188, 1974.

1233 BENIGN DUODENAL TUMORS: RADIOLOGICAL AND ENDOSCOPIC COMPARISONS. *(Fr.)* Barbier, J. P. (Laennec Hosp., Paris, France). *Sem. Hop. Paris* 50(25):1683-1689, 1974.

1234 MECKEL'S DIVERTICULUM AND ITS BLOOD SUPPLY. *(Rus.)* Starshun, G. A. (Dep. Pathol. Anat., S. M. Kirov Metall. Works, Makeenka, USSR). *Arkh. Anat. Gistol. Embriol.* 67(10):86-90, 1974.

1235 CLINICAL ASPECTS OF DIVERTICULA OF THE SMALL INTESTINE WITH THE EXCEPTION OF MECKEL'S DIVERTICULUM. *(It.)* Tagliaferri, G.; Cuccu, P.; Perozzi, A.; Preziosi, M. (S. Spirito Hosp., Sassia, Italy). *Chir. Gastroenterol.* 8(2): 214-227, 1974.

1236 INTRALUMINAL DUODENAL DIVERTICULUM: REPORT OF 2 CASES. *(Fr.)* Bories-Azeau, A.; Cabanettes, L.; Godlewski, G.; Dayan, L.; Vallat, C.; Peraldi, R. (St. Eloi Hosp., Montpellier, France). *J. Chir. (Paris)* 107(4):421-434, 1974.

1237 POSTOPERATIVE EXTERNAL DUODENAL FISTULAS. *(Rus.)* Popandopulo, G. D.; Ivaneko, S. D. (A. M. Gorky Med. Inst., Donetsk, USSR). *Vestn. Khir.* 113(9):119-124, 1974.

1238 EXTERNAL FISTULAS OF THE SMALL INTESTINE. *(Pol.)* Gacyk, W.; Dybicki, J. (Acad. Med., Gdansk, Poland). *Wiad. Lek.* 27(11):1031-1035, 1974.

1239 CLINICAL AND RADIOLOGICAL CORRELATIONS IN ACUTE OBSTRUCTION OF THE SMALL INTESTINE DUE TO STRANGULATION. *(Rus.)* Shcherbatenko, M. K.; Umanskaia, V. S. (N. V. Sklifosovskii Inst. Emerg. Med. Treat., Moscow, USSR). *Khirugiia (Mosk.)* (2): 48-52, 1974.

1240 OBSTRUCTION OF THE SMALL INTESTINE CAUSED BY A GALLSTONE. *(Pol.)* Czekalski, K.; Taniewski, W.; Przepiorski, J. (Munic. Hosp. Emerg. Surg., Warsaw, Poland). *Wiad. Lek.* 27(6):547-550, 1974.

1241 RECURRENT ILEUS DUE TO DEVELOPMENTAL ANOMALY OF THE INTESTINE. *(Pol.)* Chrzanowska, M.; Iliew, G.; Kowalski, T.; Mszanecki, J. (Dist. Hosp., Kamienna Gora, Poland). *Wiad. Lek.* 27(5): 443-445, 1974.

1242 RETROPERITONEAL RUPTURE OF THE DUODENUM. *(It.)* Interlandi, G. (Gen. Prov. Hosp., Bassano del Grappa, Italy). *Chir. Gastroenterol.* 8(1):92-103, 1974.

1243 ACUTE POSTOPERATIVE PERFORATION OF THE SMALL INTESTINE. *(It.)* Perelli, L.; Pugno, V.; Ardito, G.; Mantovani, M. (Inst. Spec. Surg. Pathol., Sacred Heart Cathol. Univ., Rome, Italy). *Policlinico* [*Chir.*] 81(2):61-68, 1974.

1244 DIAGNOSTIC AND THERAPEUTIC PROBLEMS OF TRAUMATIC PERFORATION OF THE DUODENUM IN CHILDREN. *(Ger.)* Schmidt, H. D.; Hofmann, S. (Surg. Clin., Univ. Mainz, Germany). *Bruns Beitr. Klin. Chir.* 221(5):370-378, 1974.

1245 MEGADUODENUM. A CASE REPORT AND LITERATURE REVIEW. *(E.)* Anderson, F. H. (Dep. Med., Univ. British Columbia, Vancouver, Canada). *Am. J. Gastroenterol.* 62(6):509-515, 1974.

1246 ROENTGENOCINEMATOGRAPHY DEMONSTRATING THE EFFECT OF SURGICAL SLOW-DOWN OF SMALL BOWEL TRANSIT: ANASTOMOSIS, TRANSPOSITION OF THE SMALL INTESTINE, MYOTOMY. *(Ger.)* Dux, A.; Lenz, H.; Blomer, A.; Hofer-Janker, H. (Mariahilf Hosp., Monchengladbach, Germany). *Radiologe* 14(9):409-414, 1974.

1247 THE INFLUENCE OF CORTISONE ON THE HEALING OF EXPERIMENTAL INTESTINAL ANASTOMOSES. *(Rus.)* Silvestrov, V. S. (Dep. Gen. Surg., Petrozanodsk Univ., USSR). *Eksp. Khir. Anesteziol.* (5):46-48, 1974.

1248 CLINICAL TRIALS OF SODIUM LEVORIN TABLETS SOLUBLE IN THE INTESTINE. *(Rus.)* Ashbel, S. I.; Boboslovskaia, I. A.; Khil, R. G.; Sokolova, V. G.; Filiushina, Z. G. (Gorky Res. Inst. Ind. Med. Occup. Hyg., USSR). *Antibiotiki* 19(7):645-648, 1974.

1249 DETERMINING THE LENGTH OF THE INTESTINE IN LIVING HUMAN SUBJECTS AND EXPERIMENTAL ANIMALS. *(Rus.)* Komarov, I. A.; Mokhov, E. M. (Kalinin Med. Inst., USSR). *Eksp. Khir. Anesteziol.* (6):35-38, 1974.

See also, 1114, 1136, 1137, 1165, 1166, 1167, 1168, 1169, 1170, 1171, 1270, 1274, 1305, 1306, 1307, 1336, 1476, 1511, 1518, 1526, 1536, 1546, 1547, 1555, 1562, 1595.

SMALL INTESTINE

Malabsorption

1250 NEGLECTED COELIAC DISEASE. *(E.)* McCrae, W. M.; Eastwood, M. A.; Martin, M. R.; Sircus, W. (R. Hosp. Sick Child., Edinburgh, Scotland). *Lancet* 1(7900):187-190, 1975.

One hundred and two patients in which celiac disease had been diagnosed during childhood agreed to a follow-up study. Only 2 of these patients had continued on a gluten-free diet without interruption since diagnosis. In both cases the jejunal biopsy was normal. The other 100 patients had all been eating gluten regularly and in quantity for at least one year before review. During review 55 cases of persisting celiac disease were discovered. Of these 55, 40 had stopped treatment in the mistaken belief that they had recovered from an illness which they understood to be temporary. The remaining 15 were aware of the continuing nature of the disorder, but were unable to maintain treatment without continuing dietetic advice. Symptoms in this group with continuing disease were not prominent. A few complained of lassitude, abdominal discomfort, or occasional diarrhea, but the symptoms were not compelling. Only 2 patients sought medical advice and they felt they had recovered after being treated for anemia. Clearly, untreated celiac disease had affected the growth of these patients. As a group, the 55 were significantly smaller than the normal population. Their weights were related normally to their heights and thus gave no indication of continuing malabsorption. Serum proteins and magnesium were entirely normal. No case of rickets or osteomalacia was found. The frequency of anemia was high. The findings of this study indicate that most cases of untreated celiac disease continue undetected and perhaps undetectable unless a jejunal biopsy is performed. The size of the total population, now regarded as having a pre-malignant condition, is unknown, but assumed to be large. This study also indicates that where the patients are not followed up and given continuing dietetic advice, effective treatment of celiac disease is not maintained.

1251 LIMITATIONS OF XYLOSE TOLERANCE TEST AS A SCREENING PROCEDURE IN CHILDHOOD COELIAC DISEASE. *(E.)* Lamabadusuriya, S. P.; Packer, S.; Harries, J. T. (Hosp. Sick Child., London, England). *Arch. Dis. Child.* 50(1):34-39, 1975.

The ability of the xylose tolerance test to serve as a screening procedure for celiac disease was reexamined in 54 children (aged 4 months through 16.4 yr) with suspected celiac disease. Five- and 24-hr urinary excretion rates of xylose were of no value in discriminating between patients with and without celiac disease. The 3-hr blood xylose concentration was likewise non-discriminatory. Three of the patients with subtotal villous atrophy (15.8%) and 8 of those with partial villous atrophy (61.5%) due to celiac disease had 1-hr blood xylose values which fell in the normal range. Withdrawal or reintroduction of dietary gluten caused variable effects on sequential 1-hr blood xylose levels. Thus, such manipulations of dietary gluten was generally not helpful in predicting those patients who would develop gluten-induced mucosal changes. Serious limitations of the xylose tolerance test as a screening procedure in childhood celiac disease were exposed by this study. It is suggested that the use of the urinary xylose test be abandoned in the pediatric population. The finding of a normal 1-hr blood xylose value clearly does not exclude a diagnosis of celiac disease even in young children who have never received a gluten-free diet. A clinical suspicion of celiac disease remains the most important single factor in deciding whether to perform a jejunal biopsy.

1252 COELIAC DISEASE DURING THE TEENAGE PERIOD: THE VALUE OF SERIAL SERUM FOLATE ESTIMATIONS. *(E.)* Weir, D. G.; Hourihane, D. O. (Sir Patrick Dun's Hosp., Dublin, Ireland). *Gut* 15(6): 450-457, 1974.

A group of 23 teenage celiac patients was followed at 3-month intervals as outpatients to assess their progress and monitor their ability to maintain a gluten-free diet. After a follow-up period of 4 to 6 yr, a detailed reassessment was made in the hospital on 10 patients, only 5 of whom had persevered with a gluten-free diet. There was no appreciable difference between the 2 groups in regard to physical development, height, or weight during the observation period. The jejunal mucosal histology of those patients who did not persist with a gluten-free diet remained flat although these patients appeared to have clinical remission. Those subjects who did persist with a gluten-free diet regime had a normal or near normal mucosal histology. Measurement of total proteins, serum albumin, calcium, phosphate and vitamin B_{12} showed no significant change when the patients were compared. No significant differences existed in the D-xylose or fecal fat absorption tests. The hemoglobin, corpuscular hemoglobin, and alkaline phosphatase concentrations did not significantly differ between the groups. The serum iron and folate levels were not significantly different at the beginning of the study, however, at the end of the hospitalized follow-up period, these

were the only parameters which separated the 2 groups significantly. The serum iron values were 91.4 and 43.2 mg%, and the serum folate levels were 8.6 and 2.0 ng/ml for the gluten-free and the non-gluten-free diet groups, resp. Although the serum iron values separated the 2 groups as a whole, the individual variation was such as to make it of limited practical value. The serum folate levels, on the other hand, apart from dividing the 2 groups, seemed to correlate reasonably closely with the variation in each individuals' dietary regime. The findings of this study suggest that the natural history of celiac disease is for clinical remission to occur during early adult life regardless of dietary control. This remission, however, does not pertain to jejunal mucosal histology. The study further demonstrates the importance of serial serum folate level measurements to determine the clinical progress of the patient and whether or not the subject is maintaining a gluten-free diet.

1253 MEASURING THE RESPONSE OF THE JEJUNAL MUCOSA IN ADULT COELIAC DISEASE TO TREATMENT WITH A GLUTEN-FREE DIET. *(E.)* Chapman, B. L.; Henry, K.; Paice, F.; Coghill, N. F.; Stewart, J. S. (West Middlesex Hosp., Isleworth, England). *Gut* 15(11):870-874, 1974.

Architectural changes in the small intestinal mucosa were followed for 4 to 14 months to assess the long-term response in 6 patients with adult celiac disease to treatment with a gluten-free diet. Prior to treatment, each patient had a flat or flat-with-mosaic jejunal mucosa and subtotal villous atrophy. All improved clinically, hematologically, biochemically, and histologically when treated with a gluten-free diet. Multiple jejunal diverticula were noted in 1 patient, but these were no longer present after 4 months of gluten withdrawal. The study indicated that the ratio of the areas of surface and crypt epithelium in proximal jejunal biopsies is useful for measuring the long-term mucosal response of patients to the diet. In 4 patients, this area ratio increased consistently with time on the diet after the first few weeks. In the remaining 2, the increase was not maintained. No patient achieved complete histological normality during the study. In 1 of the 3 patients followed for a year or more, the area ratio increased to within the control range, and in 2 of these 3 the ratio was about 10 times the pretreatment ratio. Overall there was a highly significant linear correlation between the area ratio and the length of time on the diet, and 60% of the variation in the ratio was explained by variation in the time. This correlation was much closer than that for surface cell height. However, the surface cell height did increase rapidly in the early weeks of treatment, while patients were still in the ward on a strict diet. The early variability of the area ratio may be due in part to errors inherent in this method of measurement, and in part to asynchronous changes in the areas of surface and crypt epithelium when gluten is first withdrawn. Thus, surface cell height was valuable for measuring the early response to treatment. Area ratio, on the other hand, was suitable for following the response over longer periods of time. It is suggested that the 2 methods of measurement are complementary.

1254 THE TOXICITY OF CERTAIN CEREAL PROTEINS IN COELIAC DISEASE. *(E.)* Cornell, H. J.; Townley, R. R. W. (R. Child. Hosp., Melbourne, Australia). *Gut* 15(11):862-869, 1974.

Gliadins from wheat, rye, and oats and from wheat glutenin were digested with pepsin, trypsin, and pancreatin and the products chromatographed on sulfopropyl Sephadex. Fractions eluted near neutral pH from wheat, rye, and oats gliadin digests all had very similar amino acid composition, although the oats fraction was higher in sulfur-containing amino acids. The major amino acids present in all were glutamine/glutamic acid and proline. The amount of fraction eluted near neutral pH from oats gliadin digest was about 13% of that eluted from digests of wheat and rye gliadins. Moreover, the yield of gliadin from oatmeal was only 0.5% compared with 2.4% and 2.8% from rye and wheat flours, resp. However, even this small amount, thought to be associated with toxicity, suggests that oats should be excluded from a diet regarded as strictly gluten-free. It is possible that there are individuals sensitive even to these low levels of toxic components. The amount of fraction eluted from wheat glutenin digest was about 70% of that obtained from wheat and rye gliadin digests. The fractions eluted near neutral pH from all protein digests were defectively digested by remission celiac mucosa. D-Xylose excretion tests with the fraction from wheat gliadin digest (fraction 9) indicated that it is harmful to subjects with celiac disease. This fraction contained several polypeptides present to the extent of about 8% of the total nitrogen applied to the column, thus providing a link between defective digestion and the toxicity of gluten in celiac disease. Pyrrolidone carboxylyl peptides did not affect D-xylose excretion, thus indicating no deficiency of pyrrolidone carboxylyl peptidase in celiac intestinal mucosa. The finding of defective digestion with counterpart fractions to fraction 9 obtained from wheat glutenin and rye and oats gliadins may be significant in the etiology of celiac disease.

1255 CELIAC DISEASE WITH PRIMARY CUTANEOUS AMYLOIDOSIS. *(E.)* Presbury, D. G. C.; Griffiths, W. A. D. (St. John's Hosp. Dis. Skin, London, England). *Br. J. Dermatol.* 92(1):109-112, 1975.

A 61 yr old man presented with an 8 week history of eczema. During the next 6 months this condition continued and lichenification of the calves was noted. He had started having explosive diarrhea 2-4 times daily, the stools being pale and bulky, soon after the onset of his skin trouble. A jejunal biopsy showed a flat mucosa typical of celiac disease. After beginning a gluten-free diet, his diarrhea stopped at once. His skin gradually improved so that over the next year the hand eczema cleared and he was left with apparent lichenification of the calves. This was made up of discrete, brown, firm, dome-shaped, rough and excoriated

papules of the type seen in lichen amyloidosus. A biopsy showed the presence of amyloid in some of the dermal papillae, using congo red, methyl violet and thioflavin-T stains, which confirmed diagnosis of lichen amyloidosus. The patient remains well, but the pruritic areas of lichen amyloidosus have not been changed by topical treatment. There is nothing to suggest that this patient has systemic amyloidosis. The skin changes have never suggested dermatitis herpetiformis, clinically or histologically. There is no obvious cause for the concurrence of the skin changes and celiac disease in this man. It is suggested that this is a first report of a combination of lichen amyloidosus and celiac syndrome.

1256 ANTIRETICULIN ANTIBODIES IN DERMATITIS HERPETIFORMIS AND ADULT COELIAC DISEASE. THEIR RELATIONSHIP TO A GLUTEN FREE DIET AND JEJUNAL HISTOLOGY. *(E.)* Lancaster-Smith, M.; Kumar, P.; Clark, M. L.; Marks, R.; Johnson, G. D. (St. Bartholomew's Hosp., Taplow, England). *Br. J. Dermatol.* 92(1):37-42, 1975.

An attempt was made to relate the occurrence of antireticulin antibodies to jejunal mucosal abnormality and the presence of gluten in the diet. Sera from 35 dermatitis herpetiformis patients (26 males and 9 females aged 17-73 yr) and 28 adult celiac disease patients (7 males and 21 females aged 19-71 yr) were examined and compared with sera from 30 normal controls. Antireticulin antibody was increased in prevalence in both dermatitis herpetiformis patients (17%) and adult celiac disease patients (45%) as compared with the control group in which the antibody was not found. In studying the relation of the antibody incidence to a gluten free diet, it is noted that 6 of 32 dermatitis herpetiformis patients on normal diets and 1 of 6 on gluten free diets had the antibody in their sera. Of the adult celiac disease patients, 9 of 12 on normal diets and 6 of 21 on gluten free diets had antireticulin antibody in their sera. Fourteen dermatitis herpetiformis patients had subtotal villous atrophy and 6 of these had the antibody in their sera, compared to only 1 of 19 with partial villous atrophy and none of 5 with a normal mucosa. Of 20 adult celiac disease patients with subtotal villous atrophy, 11 had the antibody, and of 12 with partial villous atrophy, 4 had positive tests. The 1 patient with a normal mucosa did not have this antibody. Two patterns of tissue staining were demonstrated, 1 of which was almost invariably associated with jejunal subtotal villous atrophy. In all 22 positive tests the antireticulin antibody was of the IgG class and in 14 it was also of the IgA class. There was no correlation between the immunoglobulin class of the antibody and whether the patient had dermatitis herpetiformis or adult celiac disease. Similarly there was no correlation with the type of staining pattern, dietary status or the severity of the mucosal lesion. This study confirms the relation in adult celiac disease between the occurrence of antireticulin antibody and the presence of gluten in the diet. It is recommended that screening for antireticulin antibody may be used to detect untreated patients with adult celiac disease and dermatitis herpetiformis patients with subtotal villous atrophy. The test may also aid in monitoring adherence to a gluten free diet.

1257 ASSESSMENT OF JEJUNAL FUNCTION IN PATIENTS WITH DERMATITIS HERPETIFORMIS AND ADULT COELIAC DISEASE USING A PERFUSION TECHNIQUE. *(E.)* Kumar, P. J.; Silk, D. B. A.; Rousseau, B.; Pagaltsos, A. S.; Clark, M. L.; Dawson, A. M.; Marks, R. (St. Bartholomew's Hosp., London, England). *Scand. J. Gastroenterol.* 9(8):793-798, 1974.

The jejunum of 12 patients (4 males and 8 females aged 20-60 yr) with untreated adult celiac disease and of 23 patients (18 males and 5 females aged 17-70 yr) with dermatitis herpetiformis with varying morphological abnormalities of the small bowel was perfused with a double lumen tube incorporating a proximal occlusive balloon. Nineteen normal adult volunteers with no evidence of gastrointestinal disease served as controls. Water absorption from a 56 mM solution of glucose was significantly depressed in 7 of 9 patients with adult celiac disease and in 6 of 10 with dermatitis herpetiformis with subtotal villous atrophy; actual secretion was seen in 4 patients in each group. Glucose absorption from this solution was depressed in 5 of 9 with adult celiac disease and in 4 of 10 with dermatitis herpetiformis and subtotal villous atrophy. Abnormalities of absorption were also seen in dermatitis herpetiformis patients with partial villous atrophy and with normal jejunal biopsies. Using a 35 mM saline-bicarbonate solution, bicarbonate absorption was depressed in 2 of 8 with adult celiac disease, 2 of 11 with dermatitis herpetiformis and subtotal villous atrophy, 1 of 5 with partial villous atrophy, and 2 of 5 with normal biopsies. Changes in water absorption from this solution were depressed in only 5 of 8 with adult celiac disease and 1 each with dermatitis herpetiformis and subtotal villous atrophy and a normal biopsy. It is mentioned that all but 2 patients with dermatitis herpetiformis had been on dapsone which was discontinued at least 5 days prior to the testing. To date, no evidence exists that dapsone is toxic to the jejunal mucosa, since patients may have abnormal biopsies before receiving this drug. The results obtained in this study indicate that using the perfusion technique, more abnormalities of jejunal function in dermatitis herpetiformis patients were demonstrated than would have been expected from routine gastrointestinal function tests. Also, a further link between adult celiac disease and dermatitis herpetiformis was established in that diminished absorption was seen in both conditions.

1258 GLUTEN-SENSITIVE ENTEROPATHY: SYNTHESIS OF ANTIGLIADIN ANTIBODY IN VITRO. *(E.)* Falchuk, Z. M.; Strober, W. (Peter Bent Brigham Hosp., Boston, Mass.). *Gut* 15(12):947-952, 1974.

Seven patients (2 men and 5 women, aged 9-55 yr) with gluten-sensitive enteropathy in remission were studied both immediately before and after an oral

gluten challenge lasting 6 to 12 days. Jejunal immunoglobulin (IgA and IgM) synthesis was measured in biopsy specimens obtained from the patients. The intestinal mucosa showed a striking increase in IgA and IgM synthesis. Using an affinity chromatography technique it was demonstrated that in 6 of 7 patients about half the net increase in IgA and IgM synthesis occurring after gluten challenge was attributable to the synthesis of antigliadin antibody. This study has shown that, regardless of whether serum or secretion antibody is present or absent, patients with gluten-sensitive enteropathy and not normal persons can and do produce a local antibody response to gluten. The response occurs within the intestinal mucosa where the pathological process is taking place, and it occurs early during the gluten challenge. It is suggested that the mucosal immune response may be conditioned by the presence of an immune response gene or of a cell surface receptor which facilitates a local immune response. The results from this study strengthen the hypothesis that immunological phenomena are related to the pathogenesis of gluten-sensitive enteropathy.

1259 UTILIZATION OF UREA NITROGEN FOR ALBUMIN SYNTHESIS IN THE STAGNANT LOOP SYNDROME. *(E.)* Varcoe, R.; Halliday, D.; Tavill, A. S. (MRC Clin. Res. Cent., Harrow, England). *Gut* 15(11):898-902, 1974.

The extent to which the bacterial flora make urea nitrogen available for hepatic protein synthesis was investigated in a 57 yr old male with stagnant loop syndrome. The main clinical features on presentation included severe cachexia, anasarca, the pigmented skin rash of protein malnutrition, tender hepatomegaly, and iron deficiency. Fecal fat excretion was 45 g/24 hr. Small intestinal biopsy was normal. Liver biopsy showed marked fatty infiltration. Barium meal showed partial filling and stasis of a large dilated afferent gastrectomy loop. Fluid obtained by endoscopic intubation of this loop grew *E. coli* and bacteroides. Serum and jejunal fluid contained high concentrations of unconjugated bile acids. Parenteral replacement therapy was instituted before conducting metabolic studies. Despite a smaller than normal urea pool, both the urea synthetic rate and the amount of urea metabolized in the gut were higher than in a normal control. This high non-urinary loss of urea was confirmed by recovery of only 60% of the injected labeled urea dose in the urine, whereas the control excreted 92% of the dose. The low intravascular and total exchangeable albumin pools were associated with abnormally high fractional and absolute catabolic rates of albumin in this patient. This implies an equally high synthetic rate in the steady state. ^{15}N enrichment was much higher than in the control. The decay curve for [^{15}N]albumin gave a half life of 10.3 days, considerably longer than the 7 days half life for ^{125}I-labeled albumin. Urea nitrogen was released into the gut at a rate of 4.3 g/day and this nitrogen is potentially available for protein synthesis. The relatively high enrichment by [^{15}N]albumin in this patient indicates that the liver indeed did make use of this nitrogen for albumin synthesis. Thus it is concluded that urea reutilization is increased in the stagnant loop syndrome as a result of enhanced degradation of urea by bacterial urease. It is noted that, although an increase in urea nitrogen reutilization occurs following increased bacterial urea hydrolysis, its nutritional value to the depleted nitrogen economy of the host is minimal.

1260 THE STAGNANT LOOP SYNDROME IN CHILDHOOD: REVIEW AND REPORT OF FOUR PATIENTS. *(E.)* Schreiner, R. L.; Ternberg, J. L.; Shackelford, G. D.; Keating, J. P. (Washington Univ. Sch. Med., St. Louis, Mo.). *Am. J. Dig. Dis.* 20(1):23-30, 1975.

Chronic intestinal stasis in 4 female patients (aged 5 1/2-12 yr) was studied. Three of these patients presented as adolescents with no history of bowel surgery. Each of these 3 experienced abdominal or epigastric pain and weight loss. In one, the upper gastrointestinal series disclosed a midjejunal intussusception with a lobulated mass. At laparotomy the intussusception was dilated and reduced. A section of jejunum including a hamartomatous polyp was resected. Striking dilatation and thickening of the jejunum proximal to the intussusception suggested partial obstruction of long duration. In the second adolescent, barium swallow indicated a massively dilated small bowel. Congenital ileal stenosis with markedly dilated proximal ileum was confirmed. The child's megaloblastic anemia was not studied prior to surgery because the preoperative diagnosis was lymphoma. In the third adolescent, barium study showed colonic malrotation and partial obstruction of the distal ileum at an area of marked narrowing. Exploratory laparotomy showed a volvulus involving the midileum, including a large Meckel's diverticulum. The megaloblastic anemia and low serum vitamin B_{12} in this patient were secondary to a dilated segment of ileum proximal to the chronic volvulus. Utilization of vitamin B_{12} by the bacterial overgrowth resulted in this deficiency. Differentiation of stagnant loop syndrome from neoplastic and hematologic disorders is necessary when the syndrome appears initially in later childhood with nutritional and hematologic findings dominating the clinical picture. The 5 1/2 yr old patient developed a stagnant loop syndrome as a complication of surgical repair of congenital intestinal stenosis during her first months of life. Barium examination showed marked dilatation of the duodenum and proximal jejunum associated with thickening of the mucosal folds. It is suggested that antibiotic therapy may be useful as a temporizing measure, but that the final solution of the mechanical and nutritional problems is surgical. It is suggested that long-term antibiotic therapy is not in the best interests of the pediatric patient with this syndrome.

1261 AN ELECTRON-MICROSCOPIC STUDY OF JEJUNAL MUCOSAL MORPHOLOGY IN CONTROL SUBJECTS AND IN PATIENTS WITH TROPICAL SPRUE IN SOUTHERN INDIA. *(E.)* Mathan, V. I.; Baker, S. J. (Christ. Med. Coll. Hosp., Vellore, India). *Gastroenterology* 68(1):17-32, 1975.

The ultrastructure of the jejunal mucosa obtained

by peroral biopsy from 7 control subjects and 12 patients with tropical sprue (aged 15-60 yr) from southern India was studied. Of the 12 patients, 4 had a history of 1 month or less and were from an epidemic of tropical sprue. The remaining 8 patients had endemic sprue and had been ill for periods of 4 months to 8 years. All patients had steatorrhea, low urinary xylose excretion, and defective vitamin B_{12} absorption. None of the biopsies, control or patient, could be considered normal. Compared with biopsies from control subjects in temperate zones, southern Indian control subjects showed minimal increases in lysosomes in surface epithelial cells, an occasional dark-staining degenerating epithelial cell in the upper two-thirds of villi, and an increase in cellular infiltration of surface epithelium and lamina propria. The jejunal epithelial cells of tropical sprue patients had abnormal microvilli, marked increase in lysosomes, increase in intracellular fat, and degenerative changes in rough endoplasmic reticulum and mitochondria. Two types of degenerating epithelial cells, designated pale and dark based on their staining quality, were seen in the villi. Pale staining degenerating cells were also seen in the crypts. The basement membrane region showed an amorphous deposit with many collagen fibers and fat droplets. The epithelial layer and lamina propria showed increased cellular infiltration. In one biopsy from a patient with sprue, particles of possible viral origin were noted.

1262 MALABSORPTION OF ESSENTIAL AMINO ACIDS IN TROPICAL SPRUE. *(E.)* Klipstein, F. A.; Corcino, J. J. (Trop. Malabsorption Unit, Univ. Rochester, N.Y.). *Gastroenterology* 68(2):239-244, 1975.

The absorption of 7 amino acids from an equimolar mixture containing each of the 8 essential amino acids in a concentration of 20 mM was evaluated using a marker perfusion technique in 9 patients (aged 19-70 yr) with untreated tropical sprue and in 6 healthy Puerto Rican control subjects. The absorption of isoleucine, leucine, valine, and threonine was significantly lowered in the sprue patients. In 2 patients, absorption of all 7 amino acids was within normal range. In 4 patients, values for 2 or 3 amino acids were lower than normal, and in 3 patients, values for 4 to 6 amino acids were reduced. No correlation was apparent between reduced values for amino acid absorption and the severity of intestinal abnormalities. Of the 6 neutral amino acids tested, the absorption of phenylalanine was reduced in 1 patient, methionine in 2, and the absorption of each of the other 4 neutral amino acids was reduced in either 5 or 6 patients. The absorption of the dibasic amino acid, lysine, was normal in all but 1 patient. In the 4 patients reevaluated after therapy with tetracycline (250 mg, p.o., 4 times/day) for 2 weeks, all had reduced absorption of at least 2 amino acids, and 2 patients had malabsorption of 5 or 6 amino acids. In 3 patients some evidence of improvement in intestinal function was noted with a return to normal of at least one test of absorption. Two patients had improved jejunal morphology. Of the 3 patients with evidence of improved intestinal function, the absorption of all amino acids rose to within the normal range, with the exception of methionine in 1 patient. In contrast, the 1 patient who showed no evidence of improved intestinal function, the absorption of 5 of the 6 amino acids with low pretreatment values rose, but in no instance to a normal level. Two patients with residual malabsorption of amino acids were studied again after treatment for 5 months with tetracycline (250 mg, p.o., twice/day), folic acid (1 mg/day, p.o.), and vitamin B_{12} (1000 µg, i.m., monthly). Both were asymptomatic after this prolonged treatment, gained weight, and had improved intestinal function and morphology, and absorption of all amino acids tested were within normal ranges. The results of this study indicate that the absorption of certain essential amino acids is reduced in some patients with untreated tropical sprue.

1263 BILE ACID METABOLISM AND INTESTINAL BACTERIAL FLORA AFTER THREE TYPES OF INTESTINAL SHUNT OPERATION FOR OBESITY. *(E.)* Dano, P.; Lenz, K.; Justesen, T. (Rigshosp., Copenhagen, Denmark). *Scand. J. Gastroenterol.* 9(8):767-774, 1974.

The upper jejunal bacterial flora were studied after 3 different types of intestinal shunt operation for obesity on 40 patients (27 women and 13 men, aged 15-53 yr). The results were correlated with bile acid metabolism. The patients weighed from 115 to 188 kg before operation. The types of surgery were: (I), 36 cm of the proximal jejunum measured from Trietz's ligament was anastomosed end-to-side to the distal 12 cm of the ileum measured from the ileocecal junction; (II), 24 cm jejunum was anastomosed to 24 cm ileum; (III), 12 cm jejunum was anastomosed to 36 cm ileum. Breath tests were conducted for bile acid metabolism on 13 patients prior to surgery and all results were normal. After operation, 26 of 34 patients so tested gave abnormal results. The glycine-taurine ratio was normal in all 31 patients so examined prior to operation. Postoperatively this ratio had increased in 12 of 33 patients. Prior to operation, the bile acid concentration was abnormal in 7 of 31 patients. Shunt operation was followed by an abnormally low bile acid concentration in 23 of 33 patients. The decrease in bile acid concentrations after type I and II surgery were statistically significant. Although free bile acids were not noted prior to operation, they were discovered afterwards in 1 patient who received a type II operation and in 4 who received type III surgery. The number of enterobacteria increased in the upper jejunal juice following surgery. The upper jejunal flora were particularly abnormal in patients subjected to type III surgery. Thus, it was concluded that bile acid metabolism in bypass operated patients may depend on bile acid malabsorption and partly on the abnormal intestinal flora.

1264 CALCIUM ABSORPTION AND BONE MINERAL CONTENTS FOLLOWING INTESTINAL SHUNT OPERATION IN OBESITY. A COMPARISON OF THREE TYPES OF OPERATION. *(E.)* Dano, P.; Christiansen, C. (Rigshosp.,

Copenhagen, Denmark). *Scand. J. Gastroenterol.* 9(8):775-779, 1974.

Calcium absorption and bone mineral contents were examined in 16 patients (4 men and 12 women, aged 18-49 yr) 1 to 2 years after intestinal shunt operation for obesity. Three types of operation had been used, all of them preserving 48 cm small intestine in function, but with varying jejunum:ileum ratio: Type I, 36 cm/12 cm; type II, 24 cm/24 cm; type III, 12 cm/36 cm. Calcium absorption was severely depressed, particularly in type I patients with 8% absorption against a normal of 21%. Type I and II patients had the most decreased bone mineral contents, on an average 88% of normal values. The calcium malabsorption was related to the length of distal small intestine, where passive calcium diffusion predominates, rather than to the length of functioning duodenum and jejunum, where the active calcium absorption takes place. Possibly the abnormal bile acid metabolism associated with a high jejunum:ileum ratio further increases calcium malabsorption. A longer ileal segment secured a higher calcium absorption and led to less demineralization of the bones. It is recommended that the type III operation be used for patients subjected to intestinal shunt operation for obesity.

1265 ORAL BUTTER FAT TEST MEAL WITH SERUM NEPHELOMETRY IN SUSPECTED FAT MALABSORPTION. *(E.)* Bentley, S. J.; Eastham, R. D.; Lane, R. F. (Frenchay Hosp., Bristol, England). *J. Clin. Pathol.* 28(1):80-81, 1975.

The micronephelometer was used to measure changes in serum lipids following the standard oral butter fat test meal of Kabler, Atwood and Schilling, in which a satisfactory increase in serum lipids suggests that there is no fat malabsorption. After a 10 hr fast, subjects were fed orange juice (50 ml), 2 or 3 rounds of toast with pure butter (0.5 g/kg), and 1 or 2 cups of unsweetened tea or coffee with milk, if desired. The test centered on 62 patients with gastrointestinal disorders, including 36 with diarrhea, ulcerative colitis, diverticular disease, and other disorders without overt malabsorption, 10 cases of celiac disease treated with gluten-free diet and one case not so treated, and 15 patients suffering from malabsorption. The results of the test indicated that the butter meal can be used as a preliminary test before fecal fat estimation. If the butter fat meal is followed by a reduced serum lipid increase in a patient, it would then be justifiable to place that patient on a standard diet with a known fat content, for adequate markers to be given, and a careful 3-day fecal fat estimation be carried out. Patients in whom a satisfactory increase in serum lipids of more than 20 LSI units at 2 hr was found would not require fecal fat estimation, unless otherwise clinically indicated.

1266 ABSENCE OF BRUSH BORDER SUCRASE-ISOMALTASE COMPLEX IN CONGENITAL SUCROSE INTOLERANCE. *(E.)* Schmitz, J.; Commegrain, C.; Maestracci, D.; Rey, J. (INSERM, Hosp. Dis. Child., Paris, France). *Biomedicine* 21(11):440-443, 1974.

An attempt was made to compare the brush border membrane protein pattern of a patient lacking sucrase-isomaltase activities to a normal brush border membrane protein pattern in order to gain some insight on the genetic and molecular origin of congenital sucrose intolerance. Normal brush border membrane was studied on macroscopically normal jejunal samples obtained from surgery in 3 children aged 5 months to 3 1/2 yr. Sucrase-isomaltase deficient brush border membrane was obtained from a 10 yr old patient. Polyacrylamide gel electrophoresis indicated that sucrase-isomaltase complex was lacking in the membrane from the patient. This absence may be interpreted as evidence for no protein being synthesized. Or it may also be secondary to the synthesis of an abnormal protein that could not be integrated in the brush border membrane. Contradictory results of other studies point to the possibility of different types of mutation at the origin of sucrose intolerance, as of other inborn errors of metabolism.

1267 THE GLUCOSE-GALACTOSE MALABSORPTION SYNDROME IN A 23-YEAR-OLD WOMAN. *(E.)* Hughes, W. S.; Senior, J. R. (Univ. Pennsylvania Med. Sch., Philadelphia). *Gastroenterology* 68(1): 142-145, 1975.

Intestinal perfusion studies were conducted on a 23 yr old female with the glucose-galactose malabsorption syndrome. The disease was found in the course of a clinical evaluation of malabsorption. The patient had a lifelong history of diarrhea. After ingestion of 100 g of glucose p.o. her blood sugar rose only 12 mg/dl and after 50 g galactose, her blood sugar rose 15 mg/dl, but after 50 g of fructose and 25 g D-xylose, her blood sugar rose 32 mg/dl and 43 mg/dl, resp. During the glucose tolerance test she experienced weakness, nausea, cramping abdominal pain, and profuse watery diarrhea. In her history it was noted that the diarrhea during her infancy was relieved by a formula protein milk which greatly reduced the glucose and galactose content of her diet. In intestinal perfusion studies, about 30% of fructose and 8% of the glucose and galactose solutions were absorbed. These results compare with the absorption of 38% for fructose, and 58 to 64% for glucose and galactose which is recorded in normal subjects. In this patient, the absorption of glucose and galactose was linearly proportional to glucose and galactose concentrations. In the sucrose perfusion studies, a mean of about 30% of the administered sucrose solutions was hydrolyzed under the test conditions. Fructose absorption was evident, but glucose absorption was not demonstrated during sucrose perfusion. It is believed that this is only the third adult patient discovered to have the glucose-galactose malabsorption syndrome.

1268 THE RELATIVE IMPORTANCE OF THE FACTORS INVOLVED IN THE ABSORPTION OF VITAMIN E IN CHILDREN. *(E.)* Muller, D. P. R.; Harries, J. T.; Lloyd, J. K. (Inst. Child Health, London, England). *Gut* 15(12):966-971, 1974.

The vitamin E status and ease of repletion were studied in 25 children (aged 0.1-14 yr) of whom 13 had celiac disease, 4 had intestinal lymphangiectasia, and 8 had abetalipoproteinemia. In celiac disease the mean serum vitamin E concentration was 0.39, and the mean percentage peroxide hemolysis in 9 of them was 13. The mean serum concentration for intestinal lymphangiectasia was 0.28 mg/100 ml. After giving the vitamin in doses of 0.5, 10, and 15 mg/kg/day, the serum levels increased, but normal levels were achieved only with the larger doses. In abetalipoproteinemia, serum vitamin E was undetectable before treatment. An oral load of the water-miscible preparation (120 mg/kg) had no effect. Acute i.m. loads (45 mg/kg over 48 hr and 12.5 mg/kg in a single dose) caused peroxide hemolysis to return to normal within 24 hr in both children so treated. Serum vitamin E was increased to 0.35 mg/100 ml after 5 or 6 hr, but no further rise was noted in the child with abetalipoproteinemia; whereas the levels in the other child, a victim of obstructive jaundice, continued to rise to well within the normal range. Long-term supplements of vitamin E were given p.o., about 100 mg/kg/day, to the children with abetalipoproteinemia. In all 8 children the vitamin could be detected in the serum within 1 year of starting treatment and in 5 the maximum levels were similar to those obtained after i.m. injection. The percentage hemolysis fell during vitamin E therapy in all children in whom it was estimated, and in 7 of the 8 children the minimum levels achieved during vitamin E supplementation were within the normal range. Each of the conditions studied here can represent an experimental model in which one of the intestinal transport steps involved in the absorption of vitamin E is defective. Deficiency was greatest in abetalipoproteinemia and obstructive jaundice, suggesting that normal chylomicron formation and adequate intramural bile salt concentrations are particularly important. Pancreatic enzymes, proximal mucosal integrity, and intestinal lymphatic flow appear less important. Understanding vitamin E absorption is important in treating deficiency states of the vitamin, but it is not clear that correction of low serum levels of this vitamin is of clinical benefit to children with malabsorption.

1269 IMPAIRED CALCIUM ABSORPTION IN UREMIC MAN: EVIDENCE FOR DEFECTIVE ABSORPTION IN THE PROXIMAL SMALL INTESTINE. *(E.)* Brickman, A. S.; Coburn, J. W.; Rowe, P. H.; Massry, S. G.; Norman, A. W. (VA Hosp., Los Angeles, Calif.). *J. Lab. Clin. Med.* 84(6):791-801, 1974.

Studies of intestinal absorption of calcium were carried out in 31 normal subjects, 30 patients with advanced chronic renal failure and 14 uremic patients undergoing regular treatment with hemodialysis. Absorption of radiocalcium was determined at 1, 2, 4, 6, and 25 hr after p.o. administration of the isotope with a 200 mg calcium carrier, using a modification of an external arm-counting method. This method measures what is termed, "apparent absorption", during the first 4 hr after ^{47}Ca ingestion and true absorption thereafter. In normal subjects, apparent absorption was most rapid 0 to 2 hr after ingestion of the isotope, while there was markedly impaired absorption in the patients with uremia during this same time. Between 2 and 6 hr, absorption did not differ between the patients with renal failure and the normal subjects; however, some absorption continued in the uremic patients between 6 and 25 hr, while little or no increase occurred in the normal subjects. There was no difference between ^{47}Ca absorption in patients with chronic stable renal disease and those regularly treated with hemodialysis. Administration of 1,25-dihydroxycholecalciferol (0.68 to 2.7 μg/day) for 6 to 15 days to 8 patients with renal disease caused a marked increase in calcium absorption, particularly during the 0 to 2 hr interval. These data indicate the existence of impaired intestinal absorption of calcium, with reversal of this abnormality following treatment with 1,25-dihydroxycholecalciferol. These observations are consistent with a defect in calcium transport in the proximal part of the small intestine where vitamin D may be most active in stimulating calcium transport. A defect in renal production of 1,25-dihydroxycholecalciferol may be responsible for this abnormality.

1270 JEJUNAL ABSORPTION OF BILE SALTS AFTER RESECTION OF THE ILEUM. *(E.)* Tilson, M. D.; Boyer, J. L.; Wright, H. K. (Yale Univ. Sch. Med., New Haven, Conn.). *Surgery* 77(2):231-234, 1975.

An experiment was designed to test the hypothesis that the jejunum develops increased absorptive capacity for bile salts after loss of the ileum. Everted sacs of jejunal remnants were prepared 3 months after ileectomy in Sprague-Dawley rats. There were 2 control groups of rats, 1 in which the rats underwent sham operation, and another composed of normal, unoperated rats. The mean serosal to mucosal concentration ratio of polyethylene glycol for all sacs was 1.15, indicating a small but significant net secretion of water from the everted sacs. The ratio of taurocholate for normal jejunum and sham jejunum were 1.55 and 1.52, resp., suggesting a small net absorption for those tissues. The ratios for normal ileum and experimental jejunum were 3.08 and 2.61, resp., each of which is significantly increased over the means of normal and sham operated jejunum. The results of this experiment suggest that after an interval of time the jejunum adapts to assume a role in the task of bile salt recovery. Whether the increase in the transport of bile salts is due to either an increase in the affinity of the carrier for the substrate or to an increase in the number of carrier sites cannot be determined by this study. It appears that the alterations of cellular function noted here extend the scope of adaptation considerably beyond simple morphological compensatory hypertrophy.

1271 HYPERPHAGIA IN INTESTINAL DISEASE. *(E.)* Hall, R. J. C.; Creamer, B. (St. Thomas' Hosp., London, England). *Gut* 15(11):858-861, 1974.

The cases of 6 patients (2 women and 4 men, aged 25-53 yr) with severe malabsorption and steatorrhea in whom food intake was greatly increased, are described. Caloric intake ranged from 5000 to 11000 kcal/24 hr; fat intake from 168 to 556 g/24 hr. Fecal fat excretion was elevated in 4 patients and steatorrhea was clinically present in the other 2. Despite this fecal loss, the calculated fat absorption was considerable in all patients for whom such data were available. In one patient with fecal fat excretion of 90 g/24 hr, calculated absorption of dietary fat was 84% (460 g/24 hr). The metabolic fate of this absorbed fat in patients who, despite this, remained underweight is unknown. The cause of their malabsorption was celiac disease in 3 patients, Crohn's disease with ileal resection in 2, and carcinoma of the pancreas in 1. There was no evidence of neurological or endocrine disease apart from mild diabetes mellitus in the patient with pancreatic carcinoma. However, 3 patients did suffer from severe, prolonged psychiatric depression which in 2 cases required electroconvulsive therapy. In 3 of these patients the reduction of food intake to more normal levels produced an immediate improvement in the steatorrhea. One patient maintained a constant body weight with a 40% reduction in caloric intake. Another gained weight on a low-fat diet containing half the calories he had previously consumed, but a small amount of gluten was also eliminated from the diet and this may have been responsible for the weight gain. In one patient steatorrhea diminished and weight rose during oral corticosteroid therapy and the appetite fell to normal levels. Treatment in another with pancreatin (10 g tds) resulted in decreased steatorrhea; fecal fat excretion fell from 88-20 g/day and food intake dropped from 5000 to 2000 Kcals/day. It is suggested that the association of hyperphagia and severe malabsorption and steatorrhea may be more common than realized.

1272 COELIAC DISEASE: ITS IRISH DIMENSIONS. *(E.)* McCarthy, C. F. (Reg. Hosp., Galway, Ireland). *Ir. J. Med. Sci.* 144(1):1-13, 1975.

1273 VITAMIN B_{12} ABSORPTION AND MALABSORPTION. *(Fr.)* Rey, J.; Schmitz, J. (Hosp. Sick Child., Paris, France). *Biol. Gastroenterol. (Paris)* 7(3):165-169, 1974.

1274 VITAMIN B_{12} ABSORPTION IN PATIENTS WITH ILEAL RESECTION. *(E.)* Rogers, A. C. N.; Steyn, J. H. (R. Infirm., Aberdeen, Scotland). *Br. J. Urol.* 46(6):625-629, 1974.

1275 LACTOSE AND SUCROSE MALABSORPTION IN LATIN AMERICAN UNIVERSITY STUDENTS. *(Sp.)* Pena Yanez, A.; Pena Angulo, J. F.; Paredes, G.; Romero Blasco, B.; Bermudez Garcia, J. M. (Med. Clin. B, Univ. Granada, Spain). *Rev. Esp. Enferm. Apar. Dig.* 44(3):231-248, 1974.

1276 CHARACTERIZATION OF HUMAN INTESTINAL LACTASE AND HETERO-β-GALACTOSIDASES OF INFANTS AND ADULTS. *(E.)* Lebenthal, E.; Tsuboi, K. T.; Kretchmer, N. (Div. Dev. Biol. Nutr., Stanford Univ., Calif.). *Gastroenterology* 67(6):1107-1113, 1974.

1277 ROLE OF BILE ACIDS IN FAT MALABSORPTION IN PREMATURE INFANTS. *(Ger.)* Signer, E.; Murphy, G. M.; Anderson, C. M. (no affil.). *Helv. Paediatr. Acta* 33 (suppl):23, 1974.

1278 INTESTINAL TRANSPORT OF AMINO ACIDS, GLUCOSE AND ELECTROLYTES IN A PATIENT WITH PHENYLKETONURIA. *(E.)* Wapnir, R. A.; Lifshitz, F. (North Shore Univ. Hosp., Manhasset, N.Y.). *Clin. Chim. Acta* 54(3):349-356, 1974.

1279 COPPER METABOLISM IN THE STEELY-HAIR SYNDROME. *(E.)* Lott, I. T.; DiPaolo, R.; Schwartz, D.; Janowska, S.; Kanfer, J. N. (Eunice Kennedy Shriver Cent. Ment. Retard., Waltham, Mass.). *N. Engl. J. Med.* 292(4):197-199, 1975.

1280 SELECTIVE HYPOGAMMAGLOBULINEMIA WITH PERSISTENCE OF IgE, MALABSORPTION AND A NUTRITIONALLY DEPENDENT, REVERSIBLE DEFECT IN CELL-MEDIATED IMMUNITY. *(E.)* Levin, D. M.; Lipsky, P. E.; Kirkpatrick, C. H. (Natl. Inst. Allergy Infect. Dis., Bethesda, Md.). *Am. J. Med.* 58(1):129-134, 1975.

1281 SELECTIVE MALABSORPTION OF VITAMIN B_{12}, PROTEINURIA AND HYPOGAMMAGLOBULINAEMIA — A GENETIC DEFECT. *(Ger.)* Goebell, H.; Havemann, K. (Dep. Intern. Med., Univ. Ulm, Germany). *Klin. Wochenschr.* 52(18):874-880, 1974.

1282 CAUSES OF SOME DEFICIENCIES IN GASTROINTESTINAL ABSORPTION OF PENICILLIN. *(Fr.)* Berthoud, S.; Blanc, M. H.; Rossier, J.; Fabre, J. (Med. Polyclin., Univ. Geneva, Switzerland). *Helv. Med. Acta* 27(5/6):370-371, 1974.

1283 DETECTION OF DISORDERS IN BILE SALT METABOLISM IN MALABSORPTION SYNDROMES. *(Fr.)* Voirol, M.; Borel, G. A.; Magnenat, P. (Med. Clin., Univ. Lausanne, Switzerland). *Helv. Med. Acta* 37(5/6):398, 1974.

1284 EFFECTS OF ETHANOL ON SMALL INTESTINAL ABSORPTION. *(E.)* Krasner, N.; Cochran, K. M.; Thompson, C. G.; Carmichael, H. A.; Russell, R. I. (Stobhill Gen. Hosp., Glasgow, Scotland). *Gut* 15(10):831-832, 1974.

1285 ABSORPTION ACTIVITY OF THE SMALL INTESTINE AFTER PARTIAL RESECTION OF LUNG. *(Ukr.)* Faitelberg, R. O.; Nguen-Tai-Luong (Dep. Hum. Anim. Physiol., Univ. Odessa, USSR). *Fiziol. Zh.* 20(6):773-779, 1974.

1286 EVALUATION OF INTESTINAL ABSORPTION BY THE D-XYLOSE TEST IN PATIENTS WITH BRONCHIAL ASTHMA WITH REFERENCE TO SKIN TESTS FOR FOOD ALLERGENS. *(Pol.)* Gruszecka, M. (Acad. Med., Bialystok, Poland). *Pol. Tyg. Lek.* 29(33):1425-1427, 1974.

1287 LACTOFERRIN AND IRON ABSORPTION IN THE SMALL INTESTINE. *(E.)* de Vet, B. J. C. M.; van Gool, J. (Dep. Exp. Intern. Med., Univ. Amsterdam, Netherlands). *Acta Med. Scand.* 196(5):393-402, 1974.

1288 CHRONIC MESENTERIC ARTERIAL INSUFFICIENCY CAUSED BY VASCULAR NEUROFIBROMATOSIS. *(E.)* Brunner, H.; Stacher, G.; Bankl, H.; Grabner, G. (2nd Med. Clin., Univ. Vienna, Austria). *Am. J. Gastroenterol.* 62(5):442-447, 1974.

1289 MICROBIAL COLONIZATION OF THE GASTROINTESTINAL TRACT AND DIARRHEA. *(Ger.)* Bernhardt, H.; Knoke, M. (Dep. Clin. Biol., Ernst Moritz Arndt Univ., Greifswald, Germany). *Z. Gesamte Inn. Med.* 29(15):632-635, 1974.

See also, 1114, 1129, 1139, 1221, 1222, 1430, 1431, 1532, 1565.

LARGE INTESTINE

1290 INHIBITION OF LEUCOCYTE MIGRATION IN PATIENTS WITH LARGE INTESTINAL CANCER BY EXTRACTS PREPARED FROM LARGE INTESTINAL TUMOURS AND FROM NORMAL COLONIC MUCOSA. *(E.)* McIlmurray, M. B.; Price, M. R.; Langman, M. J. S. (City Hosp., Nottingham, England). *Br. J. Cancer* 29(4):305-311, 1974.

To determine if the technique of leukocyte migration inhibition is a sensitive measure of cell-mediated immunity, the responses of leukocytes from 25 patients (mean age 63.6 yr) with large bowel cancer to extracts prepared from both large bowel tumors and normal large bowel mucosa were examined and compared with the responses obtained with leukocytes from age and sex matched control individuals (mean age 62.7 yr). In 15 experiments, cancer extract was added to the leukocytes from both cancer patients and non-cancer controls to give final protein concentrations of 1 mg/ml or 2 mg/ml, and the migration index was expressed as the ratio of the mean area of migration of leukocytes incubated with cancer extract to that of the leukocytes from the same source incubated with medium alone. In a further 10 experiments, leukocytes were incubated in both cancer and normal mucosal extract, again at protein concentrations of 1 mg/ml or 2 mg/ml and the specific migration index expressed as the ratio of the mean area of migration following incubation with cancer extract compared with that of leukocytes from the same source incubated with normal extract. Indices of less than 1 indicate inhibition by cancer extract and values greater than 1, stimulation. When leukocytes were incubated with cancer extract at a protein concentration of 1 mg/ml, the mean migration index of the cancer group was only slightly lower than that for the control group (0.76 and 0.83, resp.). In 10 of 15 paired experiments, however, the migration index of the cancer patient was less than that of the control and the differences were statistically significant. At extract protein concentrations of 2 mg/ml, the mean specific migration index in the cancer patients was 0.88 and in the control patients, 1.01. At 1 mg/ml, the mean specific migration index was 0.78 in the cancer patients and 0.87 in the controls. In 7 of 10 paired experiments at 1 mg/ml and in 7 of 10 paired experiments at 2 mg/ml, the specific migration index in the cancer patients was less than that of the corresponding controls and the differences were statistically significant. There was no correlation between the degree of migration inhibition in the cancer patients and the extent of the tumor found at operation. It is concluded that immunological reactivity can be demonstrated to tumor extracts by *in vitro* leukocyte preparations. Nonspecific toxicity cannot account for the findings since both cancer and control group samples underwent incubation with the same extracts under the same conditions. Since reactivity tended to be greater in response to cancer extracts than to normal mucosal extracts, it is further suggested that the responses are primarily directed at some component of the tumor itself.

1291 PROGNOSTIC FACTORS IN COLON CARCINOMA. CORRELATION OF SERUM CARCINOMEMBRYONIC ANTIGEN LEVEL AND TUMOR HISTOPATHOLOGY. *(E.)* Zamcheck, N.; Doos, W. G.; Prudente, R.; Lurie, B. B.; Gottlieb, L. S. (Harvard Med. Sch., Boston, Mass.). *Hum. Pathol.* 6(1):31-45, 1975.

1292 FAMILIAL POLYPOSIS AS A DEFINITE FORM OF PRECANCER. *(Ger.)* Schier, J. (Surg. Clin., Univ. Mainz, Germany). *Langenbecks Arch. Chir.* 337:772-774, 1974.

1293 PROPHYLACTIC TREATMENT WITH BACITRACIN-NEOMYCIN AND TETRACYCLINE IN SURGERY OF COLON AND RECTUM. *(E.)* Jorgensen, S. J.; Weile, F. (Kobenhavns Amts Hosp., Glostrup, Denmark). *Acta Chir. Scand.* 140(6):491-493, 1974.

1294 THE HEALING OF CHRONIC COLONIC ULCERS IN THE RAT. *(E.)* Reeve, D. R. E. (R. Coll. Surg. England, London). *Br. J. Exp. Pathol.* 55(6): 533-540, 1974.

1295 EVANESCENT COLITIS IN A CHILD. *(E.)* Friedland, G. W.; Filly, R. (Stanford Univ. Med. Cent., Calif.). *Pediatr. Radiol.* 2(1):73-74, 1974.

See also, 0817, 1137, 1170, 1171, 1177, 1526, 1541, 1547, 1584, 1595.

LARGE INTESTINE

Ulcerative Colitis

1296 ULCERATIVE COLITIS IN THE JEWISH POPULATION OF TEL-AVIV YAFO. II. THE RARITY OF MALIGNANT DEGENERATION. *(E.)* Gilat, T.; Zemishlany, Z.; Ribak, J.; Benaroya, Y.; Lilos, P. (Ichilov Hosp., Tel-Aviv, Israel). *Gastroenterology* 67(5):933-938, 1974.

Three cases of carcinoma of the colon were found among 504 Jewish patients (260 men and 244 women, aged 1 to over 70 yr) with ulcerative colitis who resided in the Tel-Aviv area. The majority of cases had substantial or universal involvement of the colon and the length of follow-up was 1-46 yr. The mean follow-up was 7.54 yr with more than 100 cases studied for more than 12 yr. The expected incidence of carcinoma of the colon for a matched group of general population would be 0.9 cases. The 3 cases found in this series amounted to an incidence of 0.6%. All 3 of these patients had been born in Europe and had resided in Israel for 11 to 44 yr. The increased incidence of carcinoma of the colon in patients with ulcerative colitis has been confirmed by numerous studies in various parts of the world. However, in most of these studies the incidence ranged from 2.5 to 7.6% or even higher. The 0.6% incidence found in this study is obviously much lower. These differences suggest that the incidence of carcinoma of the colon in ulcerative colitis patients may vary in different populations.

1297 METABOLIC ALKALOSIS IN "TOXIC MEGACOLON". *(E.)* Caprilli, R.; Alessandrini, A.; Dotta, F. (2nd Med. Clin., Univ. Rome, Italy). *Rend. Gastroenterol.* 6(1):23-28, 1974.

The acid-base balance in 8 patients with acute, severe ulcerative colitis complicated by toxic megacolon was studied. A disorder of the acid-base balance characterized by metabolic alkalosis was present in all 16 observations. The pCO_2 value was often below normal, indicating not only the absence of the compensatory respiratory mechanism, but the presence of hyperventilation as well. Hypokalemia was present in 8 observations (50%), hyponatremia in 4 (25%) and hypochloremia in 14 (87%). The mean value of Cl^- was very low (91 mEq/liter) while those of K^+ and Na^+ were within the normal range. Although hyponatremia was present in only 25% of the observations, serum Na^+ data showed a correlation with serum bicarbonate inasmuch as a very low Na^+ value corresponded to a moderate increase of bicarbonate, while the Na^+ became normal when the bicarbonate reached very high levels. This suggests that Na^+ depletion occurs very early in toxic megacolon and that the increased plasma bicarbonate levels are probably due to the secondary enhancement of renal tubular resorption of Na^+. In the presence of Cl^- depletion, there is a deficiency of resorbable anions. Therefore, tubular resorption of Na^+ occurs with a persisting removal of bicarbonate from the renal tubular fluid and in exchange for H^+ and K^+ with resulting aciduria, kaliuria, and metabolic alkalosis. Toxic megacolon must be corrected by the administration of chloride, potassium, sodium, and fluids. Minimum daily doses administered parenterally in this study were 230 mEq for sodium and chloride, 120 mEq for potassium and 2500 ml of fluids. Of these 8 patients, 7 recovered and subsequently underwent elective surgery. The 1 death occurred in a patient undergoing urgent surgery in whom a very high degree of colonic dilatation and extremely severe metabolic alkalosis were present.

See also, 1150, 1295.

PANCREAS

1298 PANCREATIC ABSCESS. *(E.)* Jones, C. E.; Polk, Jr., H. C.; Fulton, R. L. (Univ. Louisville Sch. Med., Ky.). *Am. J. Surg.* 129(1): 44-47, 1975.

A review of 13 patients with pancreatic abscess (10 men and 3 women, aged 24-64 yr) was conducted. Only one patient presented with the classic syndrome of successfully treated acute pancreatitis followed in several weeks by recrudescence of symptoms and onset of a septic course. Biliary tract disease was present in 5 patients, chronic alcoholism in 3, blunt pancreatic trauma in 2 and penetrating pancreatic trauma in 3. In general, the signs and symptoms were vague and nonspecific. Of the 8 who presented with evidence of peritonitis and occult progressive sepsis, 4 had recent histories of pancreatitis. Pancreatic abscesses after pancreatic trauma were encountered in 5 patients. Roentgenograms were helpful in preoperative diagnosis in 6 patients, and retrospectively in 2 more. Routine abdominal x-ray examinations made in 11 patients gave abnormal results in 3. Contrast studies were extremely helpful in 6 of the 8 patients so evaluated. Upper gastro-

intestinal series in 4 patients demonstrated flattening and anterior displacement of the stomach, retrogastric extraluminal air, or widening and effacement of the duodenal loop. Intravenous pyelography, showing blurred contour and lateral displacement of the left kidney with extrinsic compression on the collecting system, was diagnostically useful in 1 patient. Sinus tract injection documented pancreatic abscess in another. Barium enema and an i.v. pyelogram gave normal results in 2 patients subsequently found to have pancreatic abscess. Nine patients were treated and 8 survived. External drainage was employed in 8 and internal gastric drainage in 1 patient. The interval between hospitalization and successful diagnosis and drainage averaged 6 days in survivors. Diagnosis and external drainage were delayed 21 days in the 1 treated patient who died. Four patients were not treated and none survived. Preoperative assessment was correct in 4 patients. The majority of the abscesses were polymicrobic infections with gram-negative enteric pathogens predominating. Complications were frequent and multiple. It is concluded that successful management of pancreatic abscess requires early diagnosis and prompt external surgical drainage.

1299 THE NATURAL AND UNNATURAL HISTORY OF PANCREATIC PSEUDOCYSTS. *(E.)* Sankaran, S.; Walt, A. J. (Wayne State Univ. Sch. Med., Detroit, Mich.). *Br. J. Surg.* 62(1):37-44, 1975.

Experience with 131 pseudocysts of the pancreas in 112 patients (86 men and 26 women, aged 16-79 yr, mean age of 41 yr) is reported. The development of a pseudocyst was preceded by pancreatitis in 90% or by pancreatic trauma in 10%. Alcoholism was the principal cause of the pancreatitis. Of the 19 patients with cholelithiasis, not one stone was detected in the common bile duct. One patient was a renal transplant recipient on steroids and azathioprine. Twenty-three percent of the patients were anemic and most appeared malnourished. Radiographic studies demonstrated a mass in the region of the pancreas in only 75% of the cases. Thirty-four (26%) pseudocysts were located in the head of the pancreas. Of these, 12 (30%) caused significant obstruction of the common bile duct or duodenum. Spontaneous regression occurred in 8%. Complications arose in 33.5% of the pseudocysts, including obstruction of adjacent organs, rupture, hemorrhage and pancreatic ascites. In 14 pseudocysts, rupture into the gastrointestinal tract, the peritoneal cavity or the pleural cavity, when unassociated with hemorrhage, had a mortality rate of 14%. In 16 patients with significant hemorrhage associated with a pseudocyst, the mortality rate was 61%; early direct surgical control is advocated. Pancreatic ascites was associated with 14.5% of the pseudocysts and had a 40% recurrence rate, which can be greatly diminished when operative procedures are guided by pancreatography. The mortality rate for the surgical treatment of 97 uncomplicated pseudocysts was 6% and the recurrence rate was 11%. The prime cause of death was hemorrhage. Of the 131 pseudocysts, 7 were synchronous and 12 were metachronous. It is suggested that small cysts of the head of the pancreas, impalpable at operation but demonstrable by pancreatography, may be the cause of severe, continuing or recurrent symptoms.

1300 SPONTANEOUS RESOLUTION OF PANCREATIC PSEUDOCYSTS: IMPLICATIONS FOR TIMING OF OPERATIVE INTERVENTION. *(E.)* Bradley, III, E. L.; Clements, L. J. (Emory Univ. Sch. Med., Atlanta, Ga.). *Am. J. Surg.* 129(1):23-28, 1975.

Diagnostic ultrasound was used in the management of 38 patients with pancreatitis who presented with either a palpable abdominal mass or marked anterior gastric or duodenal displacement by upper gastrointestinal series. During this investigation, cystic masses in 5 patients (4 men and 1 woman, aged 14-43 yr) spontaneously resolved as serially documented by clinical, radiologic, and ultrasonic means. Of these 5 patients, 3 were chronic alcoholics and the other 2 suffered with diabetes. In each case the abdominal mass spontaneously disappeared within 7 days. The pseudocyst recurred in 1 patient after 4 months time. There has been no recurrence in the remaining 4 patients over a period as long as 12 months. Two of these patients have had recurrent pancreatitis with no recurrence of the pseudocyst. This report clearly demonstrates that spontaneous resolution of retrogastric cystic structures in patients with presumed pancreatic pseudocysts does occur. Since no histologic information is obtainable by the technique of ultrasound, absolute identification of the origin of the retroperitoneal cysts is not possible. However, the absence of any abnormalities on i.v. pyelography, and knowing the anatomical location in a group of patients with historical and clinical evidence of active pancreatic disease, the assumption that these cystic structures were indeed pesudocysts is not unwarranted. It is suggested that the technique of ultrasound be more widely used for definitive diagnoses which may avoid unnecessary explorative surgery.

1301 DIMINISHED EXCRETION OF BICARBONATE FROM THE SINGLE SWEAT GLAND OF PATIENTS WITH CYSTIC FIBROSIS OF THE PANCREAS. *(E.)* Kaiser, D.; Drack, E. (Dep. Pediatr., Univ. Bern, Switzerland). *Eur. J. Clin. Invest.* 4(4):261-265, 1974.

Using pH-microelectrodes the excretion of H^+ and HCO_3^- in relation to flow rate was investigated in a single sweat gland in 8 cystic fibrosis children (5 males and 3 females). In the sweat of patients with cystic fibrosis, the pH values were extremely high, significantly exceeding control values. Also in the cystic fibrosis children, the majority of the values for HCO_3^- concentration exceeded those of the control group, with the highest values amounting to 75 mEq/liter. These results indicate that, in the single sweat gland of cystic fibrosis patients in addition to increased concentrations of Na^+ and K^+ the excretion of H^+ and HCO_3^- differs from normal. A more alkaline and HCO_3^--rich sweat was produced at all sweat rates. It is suggested that, due to reduced secretion of H^+ in the duct, both the reabsorption of Na^+ and the secretion of K^+ may be impaired. The reduced flux of H^+ may similarly be responsible for the excretory defect in the duct system of the

pancreas. Reduced flux of protons across the luminal membrane of epithelial cells is proposed as a general feature of exocrine gland defects. It is then possible to describe both the cationic transport defect of the sweat gland duct and the anionic bicarbonate-secretory defect in one term.

1302 CARCINOMA OF THE PANCREAS IN A 14-YEAR-OLD BOY. *(It.)* Vincenti, R.; Chiavarelli, R. (Dep. Surg. Symptomatol., Univ. Rome, Italy). *Policlinico [Chir.]* 81(2):69-77, 1974.

1303 CRITERIA FOR LIMITING RADICAL OPERATIONS FOR CANCER OF THE PANCREATODUODENAL REGION. *(Rus.)* Linchenko, I. F.; Gumilevskaia, E. M. (Stavropol Med. Inst., USSR). *Khirurgiia (Mosk.)* (1):72-78, 1974.

1304 ACUTE MESENTERIC-PORTAL OBSTRUCTION DURING CEPHALIC DUODENOPANCREATECTOMY. THERAPEUTIC POSSIBILITIES IN TWO CASES. *(Fr.)* Clot, J. P.; Bacourt, F.; Cady, J.; Mercadier, M. (La Pitie Hosp., Paris, France). *J. Chir. (Paris)* 107(1):53-60, 1974.

1305 MORPHOLOGICAL AND HISTOCHEMICAL CHANGES IN THE STUMP OF THE PANCREATIC GLAND AFTER PANCREATODUODENAL RESECTION. *(Rus.)* Aganezov, S. A.; Perzadaev, R. O. (I.P. Pavlov 1st Med. Inst., Leningrad, USSR). *Arkh. Anat. Gistol. Embriol.* 67(10):15-19, 1974.

1306 GASTROINTESTINAL AND ENDOCRINE FUNCTION AFTER PARTIAL DUODENOPANCREATECTOMY. *(Ger.)* Mielke, F.; Beger, H. G.; Schirop, T. (Med. Clin., Free Univ. Berlin, Germany). *Dtsch. Med. Wochenschr.* 31(5):171-176, 1974.

1307 BLOOD VESSELS OF THE PANCREATODUODENAL REGION AFTER EXPERIMENTAL RESECTION OF THE PANCREAS. *(Rus.)* Poliak, R. I.; Khunov, Z. D. (Stavropol Med. Inst., USSR). *Arkh. Anat. Gistol. Embriol.* 67(10):91-96, 1974.

1308 TOTAL PANCREATECTOMY IN JAPAN. *(Jpn.)* Nakase, A. (Kyoto Univ. Med. Sch., Japan). *Arch. Jpn. Chir.* 42(2):151-156, 1973.

1309 EXOCRINE PANCREATIC FUNCTION IN RENAL INSUFFICIENCY. *(Ger.)* Gerhardt, W.; Stein, G.; Bosseckert, H.; Graupner, C.; Noske, A. (Med. Clin., Friedrich Schiller Univ., Jena, Germany). *Z. Ges. Inn. Med.* 29(21):895-898, 1974.

1310 CONSERVATIVE TREATMENT OF MECONIUM ILEUS WITH VISOTRAST ENEMAS. *(Ger.)* Hormann, D.; Gottschalk, M. (Clin. Pediatr. Surg., Karl Marx Univ., Leipzig, Germany). *Kinderaerztl. Prax.* 42(3):106-112, 1974.

See also, 1128, 1140, 1141, 1168, 1172, 1173, 1175, 1176, 1203, 1271, 1316, 1317, 1414, 1502.

PANCREAS

Pancreatitis

1311 ACUTE PANCREATITIS AFTER ENDOSCOPIC/RADIOLOGICAL PANCREATICOGRAPHY (ERP). *(E.)* Ruppin, H.; Amon, R.; Ettl, W.; Classen, M.; Demling, L. (Med. Dep., Univ. Erlangen-Nuremberg, Erlangen, Germany). *Endoscopy* 6(2):94-98, 1974.

A retrospective examination of the period following endoscopic-radiological pancreaticography in 569 patients is reported. In all of the 569 cases, 60% contrast medium had been used. Subsequent to the examination, 42 patients presented symptoms of acute pancreatitis, including fever, elevated amylase in the serum or urine, leukocytosis and usually epigastric pain. An additional 6 patients presented with pseudocyst infections. An additional 188 patients showed slight signs of infection but no unequivocal indications of pancreatitis. Acute pancreatitis occurred much more frequently in patients in whom contrast medium had pervaded the parenchyma of the pancreas. On the other hand, an increase in the occurrence of pancreatitis in pathological findings of the pancreatic duct system was not noted. In 40 cases conservative therapy with antibiotics, and in severe cases additional treatment with trasylol, atropine, fasting and shock prophylaxis, produced rapid disappearance of the symptoms and a normalization of the objective findings. However, 2 fatalities occurred consequent upon acute tryptic and suppurating pancreatitis, resp. A 3rd patient died during the course of a secondary infection of a pseudocyst of the pancreas subsequent to pancreaticography. This study showed that the development of acute pancreatitis is a much more frequent complication of endoscopic-retrograde pancreatography, 7.4%, than had previously been thought. It is stressed that the filling of the acini and the pancreatic tissue with contrast material must be avoided.

1312 THE INFLUENCE OF GLUCAGON ON ACUTE EXPERIMENTAL PANCREATITIS IN THE RAT. *(E.)* Lankisch, P. G.; Winckler, K.; Bokermann, M.; Schmidt, H.; Creutzfeldt, W. (Dep. Med., Univ. Gottingen, Germany). *Scand. J. Gastroenterol.* 9(8):725-729, 1974.

The effects of glucagon on the course of experimental acute pancreatitis induced by corn oil or by Na-taurocholate in the male Wistar rat were studied. Glucagon suspended in corn oil given s.c. at single doses of 0.24 mg every 6 hr for 18 hr, decreased pancreatic enzymes to levels between 10.9 and 38.8% of controls, and affected lipase least and phospholipase A most. Glucagon did not influence the mortality rate in severe (oil-induced) pancreatitis when given before or after operation. When rats were sacrificed at 6 hr after induction of oil-induced pancreatitis, the amount of ascites seen was significantly lower in the pretreated group. Ascites was not lowered in rats treated after induction of pancreatitis. Glucagon did somewhat lower the mortality rate in medium severe (Na-taurocholate-induced) pancreatitis both when given before and after pancreatitis induction. Except for a reduction of zymogen granules, glucagon had no significant effect on any of the morphological parameters (ascites, fat necrosis, pancreas necrosis, edema, and leukocyte infiltration). The effect of glucagon pretreatment on the increase of amlyase and lipase was investigated 6 hr after induction of oil-induced pancreatitis. This increase of both enzymes was significantly less after glucagon pretreatment. Glucagon treatment immediately postoperatively also had an effect on serum enzymes. Lipase was significantly lower in the glucagon-treated group when compared with non-treated rats 6 hr after induction of pancreatitis. In the same animals, serum amylase levels were also lowered under treatment, but not significantly so. It is thus concluded that glucagon pretreatment and treatment significantly reduced serum enzymes but did not significantly influence the mortality rate and morphological parameters of a severe or medium severe form of experimental pancreatitis.

1313 SOME CLINICAL SIGNS OF CHRONIC PANCREATITIS. *(Rus.)* Volosevich, N. V. (Goriachii Kliuch Sanit., USSR). *Vrach. Delo* (12):85-87, 1974.

1314 102 CASES OF CHRONIC PANCREATITIS: STUDY OF CLINICAL FEATURES AND COURSE. *(Fr.)* Bernades, P.; Callet, B.; Dupuy, R. (Beaujon Hosp., Clichy, France). *Nouv. Presse Med.* 4(3):149-152, 1975.

1315 PARAPANCREATITIS AS AN IMPORTANT COMPONENT OF SEVERE FORMS OF ACUTE PANCREATITIS. *(Rus.)* Filin, V. I.; Spasskaia, M. G.; Zharenkova, V. D. (I. I. Dzhanelidze Inst. Emerg. Med. Treat., Leningrad, USSR). *Vestn. Khir.* 113(9):7-13, 1974.

1316 STATISTICAL ANALYSIS OF PAIN IN PANCREATIC DISEASE. *(Fr.)* Cornee, J.; Durbec, J. P.; Yoro, M.; Lauret, F. (Res. Unit Gastrointest. Dis., INSERM, Marseilles, France). *Arch. Fr. Mal. App. Dig.* 63(5):375-384, 1974.

1317 RARE INDICATIONS FOR DUODENOPANCREATECTOMY. *(Ger.)* Hoffmann, E.; Gebhardt, C.; Derra, Jr., E. (Surg. Clin., Univ. Dusseldorf, Germany). *Bruns Beitr. Klin. Chir.* 221(5):379-385, 1974.

See also, 1142, 1174, 1178, 1304, 1481.

LIVER AND BILIARY TRACT

1318 CLINICAL USE OF CORTICOSTEROID AND IMMUNOSUPPRESSIVE DRUGS IN LIVER DISEASE. *(E.)* Fung, W. P. (Fac. Med., Univ. Singapore). *Drugs* 8(2):139-145, 1974.

The current status of the clinical use of steroids and immunosuppressants in liver disease is reviewed. Although steroids and immunosuppressants are not routinely indicated in cases of uncomplicated viral hepatitis, steroids may be used in the uncommon case complicated by: 1) prolonged cholestasis past 4 weeks in which extrahepatic causes have been excluded (prednisolone 10 mg 3 times daily until recovery); 2) chronic hepatitis persisting more than 3 months; 3) fulminant hepatitis with hepatic coma (200-500 mg hydrocortisone i.v. every 6 hr as a last resort) although results have not been encouraging. Immunosuppressants such as azathioprine and 6-mercaptopurine are not indicated in these conditions and may, indeed, aggravate the situation. Chronic active hepatitis, which carries a relatively poorer prognosis, may respond symptomatically and with reduced early mortality to prednisolone (20 mg 3 times daily until improvement then 5 mg 3 times daily maintenance) and/or azathioprine (25-50 mg daily increasing to 100 mg daily as needed). These drugs do not change overall survival or prevent cirrhosis in these patients. Steroids are of little or no value and immunosuppressants are not indicated in patients with drug-induced cholestasis, cryptogenic cirrhosis or extrahepatic obstructive jaundice. Steroids may be used in active post-necrotic or post-hepatic cirrhosis and in cholangiohepatitis complicated by *E. coli* septicemia.

1319 RADIOIMMUNOASSAY OF α-FETOPROTEIN IN HUMAN SERUM. CLINICAL VALUE IN PATIENTS WITH LIVER DISEASES. *(E.)* Chayvialle, J. A. P.; Touillon, C.; Crozier, C.; Lambert, R. (INSERM, Edouard-Herriot Hosp., Lyon, France). *Am. J. Dig Dis.* 19(12):1102-1110, 1974.

A radioimmunoassay has been developed for human α_1-fetoprotein. The method involves competitive inhibition of the binding of purified ^{125}I-α_1-fetoprotein to specific antiserum by human test serum. The sensitivity of the assay is 0.5-1.0 ng/ml of serum. Mean serum α_1-fetoprotein level in 136 healthy adults was 3.8 ng/ml. All 34 patients with histologically proven primary hepatocellular carcinoma had abnormal-

ly high levels which were, however, only moderately high (<500 ng/ml) in 5. Four of 19 patients with metastatic liver disease and 39 of 118 patients with nonneoplastic liver diseases had moderately elevated serum α_1-fetoprotein levels ranging from 7.7 to 500 ng/ml indicating that a moderately elevated level is not diagnostic for malignant hepatoma. In 1 patient with familial hemochromatosis, a transient rise (69.2 ng/ml) in serum α_1-fetoprotein was seen which subsequently returned to a stable upper-normal level. Thus, the radioimmunoassay for α_1-fetoprotein may be useful in following patients with nonneoplastic liver disease who have a high risk of developing hepatocellular carcinoma.

1320 α-FETOPROTEIN: EXPRESSION IN HUMAN DISEASE AND IN RAT EXPERIMENTAL MODELS. *(E.)*
Wepsic, H. T.; Sell, S. (San Diego Med. Sch., Univ. Calif.). *Prog. Exp. Tumor Res.* 19:297-324, 1974.

Recent observations on the relationship of α_1-fetoprotein to human malignancy and other disease states and comparison of these to findings in experimental rat tumor models are summarized. Results based on radioimmunoassay indicate that normal humans have basal levels of α_1-fetoprotein (1.5-16.5 ng/ml) which are immunologically indistinguishable from the α_1-fetoprotein associated with hepatoma. Thus, a normal limit of 20 ng/ml is generally accepted. Factors which can influence serum α_1-fetoprotein levels include race (higher in non-Caucasians), age (higher correlation with hepatoma in younger patients), sex (males>females), differentiation and size of tumor (more commonly present with poorly differentiated, large tumors) and site (most common with hepatocellular carcinoma). Studies both in Morris hepatoma-bearing and tumor-excised rats and in hepatoma-bearing humans show a correlation between serum α_1-fetoprotein concentration and the rate of growth and amount of tumor tissue present. A fall in α_1-fetoprotein concentration to normal or negligible levels following surgery is associated with removal of the tumor and may be used to follow the patient clinically. The presence of metastatic tumor in rats which had their primary excised could be predicted by rising serum α_1-fetoprotein levels up to 30 days prior to their detection on physical examination. A fall in serum α_1-fetoprotein levels has also been correlated with a clinical response to chemotherapy in humans. α_1-Fetoprotein has also been observed in patients with testicular and ovarian germinal cell tumors (especially poorly differentiated), in non-gonadal teratomas and in a few cases of tumor metastatic to the liver from lung, pancreas, stomach, gallbladder, lymphoma, urinary bladder and uterus. Transiently elevated serum α_1-fetoprotein concentrations have been associated with acute viral hepatitis; the possible relationship between α_1-fetoprotein, Australia antigen and hepatoma is still under debate. Elevated α_1-fetoprotein levels have also been detected in patients with cirrhosis, especially the postnecrotic type, in patients with regenerating liver following partial hepatectomy and rarely in patients with other disease affecting the liver. Increased α_1-fetoprotein may also be seen in the serum of pregnant females in association with fetal distress, intrauterine death or severe dysfunction.

1321 α_1-ANTITRYPSIN DEFICIENCY AND LIVER DISEASE IN CHILDHOOD: GENETIC, IMMUNOCHEMICAL, HISTOLOGICAL, AND ULTRASTRUCTURAL DIAGNOSIS. *(E.)*
Ward, A. M.; Underwood, J. C. E. (Hallamshire Hosp., Sheffield, England). *J. Clin. Pathol.* 27(6):467-472, 1974.

The clinical characteristics of 8 children with liver disease associated with α_1-antitrypsin deficiency and detailed histologic examination of liver tissue from 3 of these are presented. All the children presented initially with gradual onset of painless obstructive jaundice at 2-3 weeks of age which persisted for a few weeks before spontaneously regressing. There was no associated fever or failure to thrive and the only physical finding was an enlarged, often firm, liver. All exhibited the homozygous deficiency state characterized by serum α_1-antitrypsin levels 0.50 mg/liter and Pi phenotypes ZZ. All parents were heterozygous MZ with intermediate α_1-antitrypsin levels. All children's sera were Australia antigen- and antibody-negative. The dominant liver change seen on light microscopy was lymphoreticular infiltration and expansion of the portal tracts by fibrous proliferation. Numerous PAS-positive, diastase-resistant granules were present within the cytoplasm of hepatocytes at the periphery of the surviving lobules or regeneration nodules, but were not seen in biopsies from children with hepatitis or biliary tract obstruction in the absence of α_1-antitrypsin deficiency. Immunofluorescent microscopy using specific antisera confirmed the presence of excessive amounts of α_1-antitrypsin within hepatocytes of the diseased deficient children. Electron microscopy showed numerous 3 μm diameter globules containing amorphous material and bounded by a single membrane in apparent continuity with the endoplasmic reticulum of the periportal hepatocytes. These findings suggest that α_1-antitrypsin synthesis in the Pi ZZ homozygote is defective and that this induces, as a secondary phenomenon, an abnormality in intracellular transport.

1322 HEPATOCELLULAR CARCINOMA AND PARTIAL DEFICIENCY OF ALPHA-1-ANTITRYPSIN (MZ). *(E.)*
Rawlings, Jr., W.; Moss, J.; Cooper, H. S.; Hamilton, S. R. (Johns Hopkins Univ. Sch. Med., Baltimore, Md.). *Ann. Intern. Med.* 81(6):771-773, 1974.

Two case histories are presented in which hepatocellular carcinoma was associated with the heterozygous (MZ) state for α_1-antitrypsin deficiency. Both patients presented with liver disease of uncertain etiology. The first, a 66-yr-old male with a past history of surgically cured squamous cell carcinoma presented with ascites, hepatomegaly and edema. Serum electrophoresis showed a low α_1-globulin fraction of 0.1 g% (normal = 0.1-0.4 g%). Immunoelectrophoresis revealed an MZ phenotype. α_1-Fetoprotein was negative. The patient subsequently died of massive upper gastrointestinal bleeding and sepsis. At necropsy, the patient was found to have hepatic cirrhosis with a multifocal, nonmetastatic hepatocellular carcinoma. The hepatocytes contained PAS negative, diastase-resistant granules which were not present in tumor cells. The second patient, a 64-

yr-old white male, presented with malaise, weakness, jaundice and hepatomegaly. Serum α_1-globulin was 0.2 g%. α_1-Fetoprotein was negative and the patient had an MZ phenotype. The patient died of upper gastrointestinal bleeding and hepatic failure. Necropsy revealed hepatocellular carcinoma with severe focal bullous emphysema and metastases in the lungs. Periportal hepatocytes, but not tumor cells, contained PAS positive, diastase-resistant granules. The rarity of both the Z allele and hepatocellular carcinoma suggest a real association between the heterozygous state of α_1-antitrypsin deficiency and this malignancy.

1323 LIVER CELL CARCINOMA DURING THE PRIME OF LIFE. *(E.)* Mabogunje, O.; Rosen, P. P.; Fortner, J. G. (Mem. Sloan-Kettering Cancer Cent., New York, N.Y.). *Surg. Gynecol. Obstet.* 140(1):75-80, 1975.

The clinical and pathologic conditions as well as the response to treatment were studied in 43 patients (11-40 yr old) who were diagnosed between 1949 and 1972 as having primary liver cell carcinoma. Nineteen patients were male and 24 were female. Presenting symptoms included weight loss or right upper quadrant pain (65%), fatigue, anorexia and intermittent fever (33%) and abdominal mass (20%). Only 1 patient had jaundice. Hepatomegaly was present in 88%, being tender in about half of these. Most patients had a classic liver profile with normal bilirubin and elevated alkaline phosphatase ranging from 58-1750 IU (normal = 31-82), 5'nucleotidase (12.2-365 Cample U; normal = 3.2-13.6) and sodium sulfobromophthalein retention (2-37%; normal = 0-5%). Serum α_1-fetoprotein was present in 5 of 9 patients tested. Ten patients had angiography and 13 had a liver scan; both studies were done in 8 patients, agreeing and accurately localizing the tumor in 6 cases (75%). Two patients also had erythrocytosis, 1 developed preterminal hypoglycemia and 1 had hypercholesterolemia with hyperproteinemia. Autopsy of 13 patients revealed cirrhosis in 3. Only 3 patients had a previous history of liver disease including hepatitis and idiopathic jaundice. Tumor was confined to 1 lobe in 7 patients and both lobes in 10. Ten patients showed local nodal and 16 showed widespread metastatic disease. Primaries were multicentric in 27. Survival ranged from 3 months in 6 patients who were not treated, 1-30 months (median of 9 months) in 12 patients who received radiation only to the liver (3000-4000 rads), to 1-26 months (median of 5 months) in patients treated with chemotherapy. Of 10 patients treated by lobectomy, 2 are alive after 25 and 21 months, resp., the latter free of disease, and 2 have survived 12.5 and 13.5 yr, resp. (20% 10 yr survival). These survival rates do not differ significantly from those reported for older populations.

1324 PRIMARY HEPATIC MALIGNANCY IN PREGNANT WOMEN. *(E.)* Purtilo, D. T.; Clark, J. V.; Williams, R. (Armed Forces Inst. Pathol., Washington, D.C.). *Am. J. Obstet. Gynecol.* 121(1):41-44, 1975.

The occurrence of 2 cholangiocarcinomas and 2 hepatocellular carcinomas in 4 women (aged 25-37 yr) is described. Three died while pregnant and another died 2 months postpartum. The clinical signs and symptoms seen in these 4 patients were similar to manifestations of other diseases of the liver occurring during pregnancy. Even so, antemortem diagnoses were obtained in 3 of the cases. In the postpartum patient, radiographic evidence of metastases to the lungs, a positive pregnancy test, and carcinoma in a hepatic biopsy specimen led to the erroneous clinical and pathologic diagnosis of metastatic cholangiocarcinoma and a panhysterectomy was performed. It is urged that the physician be aware that other malignant neoplasms besides trophoblastic neoplasia may produce chorionic gonadotropin. In normal pregnancies, maternal serum contains 53 to 500 ng/ml alpha-fetoprotein (AFP), and with fetal death *in utero* this level may rise to 9 µg/ml. The finding of 20 to 25 µg/ml AFP in the serum of 1 patient, in the absence of intrauterine death, indicated a diagnosis of hepatocellular carcinoma rather than feto-maternal passage of AFP. The occurrence of primary hepatic malignancy in the 4 pregnant women does not negate the potential role of androgenic steroids in hepatocarcinogenesis, but these occurrences are contrary to the observation that liver-cell carcinoma requires an androgenic environment. Cirrhosis was present in only 1 of these patients; none was American or British white. Tests for HB-Ag and antibody were performed in only 1 patient, and the serum was positive for the HB antibody. However, this patient had previously received blood transfusions 2 yr prior to the appearance of hepatocellular carcinoma, which may be the explanation. It is suggested that the incidence of pregnancy in one of these 4 women, with its resultant immunological surveillance suppression, may have allowed the tumor-host balance in her to be tipped so that a hepatocellular carcinoma which had been dormant for 11 yr, could progress. However, it is also suggested that this could have been an entirely new tumor.

1325 EICOSATRIENOIC ACID ω9 IN SERUM LIPIDS OF PATIENTS WITH HEPATOCELLULAR CARCINOMA. *(E.)* Okazaki, N.; Araki, E. (Natl. Cancer Cent. Hosp., Tokyo, Japan). *Clin. Chim. Acta* 53(6237):11-21, 1974.

Serum lipids were analyzed for eicosatrienoic acid ω9 (20:3ω9) content in 24 patients with hepatocellular carcinoma, 14 with metastatic liver carcinoma, and 10 with liver cirrhosis. Eicosatrienoic acid ω9 was identified through the use of argentation thin-layer chromatography followed by hydrogenation and a combination of gas liquid chromatography and mass spectrometry of the trimethylsilyoxy derivatives of the methyl esters. Sera from 9 patients in the 1st group were positive for 20:3ω9 (37.5%). This fatty acid was not detected in the sera of patients with metastatic liver carcinoma, cirrhosis, or healthy controls. Sera positive for eicosatrienoic acid ω9 were also positive for α_1-fetoprotein (47.4%). No eicosatrienoic acid ω9 was found in the sera negative for α_1-fetoprotein. The acid was also observed in the primary liver tumor and the tumor metastatic to the lung in 1 patient with hepatocellular carcinoma. There are differences between the fatty acid composition of the liver and that of the lung, but the fatty acid composition was identical in both tumors when 20:3ω9 was present.

There was no deficiency of essential fatty acids in those cases that were positive for eicosatrienoic acid ω9. Essential fatty acid components (percentage values) of serum lipids were significantly higher in the 20:3ω9-positive group than in the negative cases, and the positive cases had no marked essential fatty acid deficiency. The deficiency may not be the factor causing the appearance of 20:3ω9 in serum of patients with hepatocellular carcinoma. A local deficiency may be a factor leading to the presence of 20:3ω9 in rapidly growing tumor tissue. Eicosatrienoic acid ω9 was observed only in tumor tissue; it was not present in non-invasive cirrhotic liver, lung, spleen, heart muscle, skeletal muscle, pancreas, intestine, kidney and adipose tissue. The limited distribution of this fatty acid suggests the presence of altered fatty acid metabolism and the synthesis of eicosatrienoic acid ω9 in tumor tissue of hepatocellular carcinoma.

1326 GASTRIC CANNONBALLS: A ROENTGEN SIGN OF HEPATIC METASTASES. A CASE REPORT. *(E.)* Doss, J. C.; Ferrucci, Jr., J. T. (Massachusetts Gen. Hosp., Boston). *Gastroenterology* 67(3):519-520, 1974.

A case history illustrating an unusual appearance of multiple cannonball-like defects on the stomach during conventional barium meal study is presented. The patient was a 74-yr-old Caucasian female who was well until 4 weeks before hospitalization when she began to experience intermittent episodes of nausea and vomiting. Preliminary plain film study of the abdomen showed enlargement of the liver extending into the left upper quadrant. There were multiple persistent rounded filling defects within the body of the stomach. A subsequent 99^{m} technitium hepatic scan showed marked hepatomegaly and multiple discrete space-occupying defects consistent with metastatic disease. The patient died 14 days after hospital admission. Postmortem examination revealed that the liver weighed 3100 g and contained innumerable metastatic nodules of undifferentiated carcinoma. Several tumor nodules had invaded the serosa of the stomach, thereby fixing that organ to the liver and several other nodules projected into the gastric wall without frank fixation. Undifferentiated carcinoma was also found within the left kidney. Contrary to most cases of abnormal liver disease, in this instance, a silent primary tumor, occult even at necropsy, allowed progressive liver involvement requiring hospitalization only 2 weeks before death.

1327 CASE REPORT. HEREDITARY RECURRENT CHOLESTASIS WITH LYMPHOEDEMA -- TWO NEW FAMILIES. *(E.)* Aagenaes, O. (Univ. Hosp., Oslo, Norway). *Acta Paediatr. Scand.* 63(3):465-471, 1974.

The clinical course and liver histology of 2 new cases of hereditary, neonatal cholestasis with primary lymphoedema at birth or later are described. The 2 patients were unrelated. Both had neonatal intrahepatic cholestasis, in 1 lasting for at least 10 yr, in the other only about 6 months. Lymphoedema was severe in the first case and more moderate in the latter. Liver histology showed giant cell transformation in infancy and some fibrosis or cirrhosis in later childhood. In the first family, 3 of 6 siblings were similarly affected at birth, and in the other family, the parents were related, supporting an earlier theory of an autosomal, recessive heredity as the etiology of this condition.

1328 MONOHYDROXY BILE ACIDS IN PLASMA IN INTRAHEPATIC CHOLESTASIS OF PREGNANCY. IDENTIFICATION BY COMPUTERIZED GAS CHROMATOGRAPHY-MASS SPECTROMETRY. *(E.)* Back, P.; Sjovall, J.; Sjovall, K. (Dep. Chem., Karolinska Inst., Stockholm, Sweden). *Med. Biol.* 52(1):31-38, 1974.

A detailed study of bile acids in plasma from normal pregnant women and from pregnant women with intrahepatic cholestasis was conducted using a method involving rapid computer evaluation of gas chromatographic-mass spectrometric analysis of sulfated and non-sulfated conjugated bile acid fractions. Total cholic, chenodeoxycholic and deoxycholic acids from pooled plasma samples from 30 healthy pregnant females were 1.10, 0.73 and 0.39 μg/ml, resp. Comparable values calculated from a pooled plasma sample from 8 pregnant patients with cholestasis were elevated at 7.90, 3.60 and 1.40 μg/ml, resp. Most of the bile cholic, chenodeoxycholic and deoxycholic acids were present in the non-sulfated fraction. In addition, the sulfated fraction contained lithocholic (0.19-0.41 μg/ml), 3β-hydroxy-5-cholenoic (0.06-1.28 μg/ml) and ursodeoxycholic (0.026 μg/ml) acids. Of these, only lithocholic acid (0.05 μg/ml) was found in pooled plasma from healthy pregnant females. The highest lithocholic acid concentration (0.65 μg/ml) was present in a woman without cholestasis who later gave birth to a child who developed severe transient neonatal jaundice of unknown etiology. It thus seems possible that the concentrations of lithocholic and 3β-hydroxy-5-cholenoic acids in maternal plasma are partly determined by the functional state of the fetal liver.

1329 OCCURRENCE OF HEPATIC IMPAIRMENT IN WOMEN JAUNDICED BY ORAL CONTRACEPTIVES AND IN THEIR MOTHERS AND SISTERS. *(E.)* Dalen, E.; Westerholm, B. (Natl. Board Health Welfare, Stockholm, Sweden). *Acta Med. Scand.* 195(6):459-463, 1974.

A questionnaire survey was conducted in Sweden to determine whether women with jaundice from oral contraceptives, and their mothers and sisters, differed from control women who had used oral contraceptives for at least 2 yr without adverse reactions and their mothers and sisters. On receipt of the completed questionnaires, the patients and controls were matched so that the parity was the same as was the yr of birth ± 2 yr. This matching operation produced 129 matched pairs. The patients had experienced pruritus and jaundice during pregnancy more often than the controls. The patient group also showed a higher incidence of hepatitis, gallstone symptoms and cholecystectomy. The patients' mothers had experienced pruritus during pregnancy to a greater extent than their controls. The patients' sisters showed a significantly higher incidence of pruritus during pregnancy and oral contraceptive treatment than their controls. Gallstone

symptoms and cholecystectomy were also found more often in the group of the patients' sisters. The results support the hypothesis than genetic factors play a part in the occurrence of jaundice during oral contraception and late pregnancy. The investigation also indicates that patients with jaundice due to oral contraceptive treatment must be considered as high risk patients with regard to their proneness to develop gallbladder disease.

1330 FACTORS INFLUENCING MORTALITY IN THE TREATMENT OF PYOGENIC HEPATIC ABSCESS. *(E.)* Pitt, H. A.; Zuidema, G. D. (John Hopkins Hosp., Baltimore, Md.). *Surg. Gynecol. Obstet.* 140(2):228-234, 1975.

Eighty cases of pyogenic abscess, observed over a period of 21 years, were reviewed to better define factors associated with mortality. It was noted mortality jumped from 57% to 81% as age of 70 yr was passed. Laboratory data associated with high mortality were elevated bilirubin, or SGOT levels, albumin levels less than 3.0 g%, and polymorphonuclear neutrophil leukocyte levels over 80%. Mortality was higher (88%) in patients with multiple abscesses than in those with single abscesses (31%), and significantly higher in cases of biliary origin. Another possible factor in high mortality was the presence of multiple organism infections, although the results were not significant. Of 39 patients receiving no or partial treatment (either antibiotic or surgical) 97% died, 20% of them before therapy could be given. In the 41 patients receiving both antibiotic and surgical treatment, 34% died. Seventy-one percent of these deaths had multiple abscesses. Complications, especially those accompanied by bacteremia, raised mortality. It was also noted that over the 21 yr the percentage of women and of older patients had increased. Factors influencing mortality were thus: age over 70 yr, multiple abscesses, biliary origin, associated malignancy, elevated SGOT level, hypoalbuminemia, polymorphonucleocytosis, bacteremia, aerobic infections, and pulmonary, peritoneal, or other significant complications.

1331 REYE'S SYNDROME IN THREE ISRAELI CHILDREN. POSSIBLE RELATIONSHIP TO WARFARIN TOXICITY. *(E.)* Mogilner, B. M.; Freeman, J. S.; Blashar, Y.; Pincus, F. E. (Barzilai Med. Cent., Ashkelon, Israel). *Isr. J. Med. Sci.* 10(9):1117-1125, 1975.

Three fatal cases of Reye's syndrome were studied to establish the possible etiological importance of 3-(α-acetonylbenzyl)-4-hydroxycoumarin (Warfarin) used as a rodenticide and an anticoagulant. All 3 (a 3 yr old boy and girl and a 2 yr old boy) displayed the clinical symptoms of the syndrome including "coffee-ground vomiting," clouding of consciousness and convulsions. Autopsy findings confirmed the diagnosis and showed fatty degeneration of the liver and kidney and, in at least 1 case, the myocardium. There was also brain edema with no specific inflammation. In 2 of the children, the hydroxycoumarin was definitely implicated. Two were from the same village, but no toxicological studies were made. It is concluded that Warfarin may be a new etiological factor in Reye's syndrome.

1332 NEW OBSERVATIONS IN THE CLINICAL SPECTRUM OF THE BUDD-CHIARI SYNDROME. *(E.)* Schramek, A.; Better, O. S.; Brook, J. G.; Alroy, G. G.; Gellei, B. (Rambam Gov. Hosp., Haifa, Israel). *Ann. Surg.* 180(3):368-372, 1974.

Three case histories are used to present some observations on the natural history, surgical management, and metabolic phenomena in Budd-Chiari syndrome. In one patient side-to-side portacaval shunt brought improvement with asymptomatic follow-up for 5 yr. The patient died of causes unrelated to Budd-Chiari syndrome. At postmortem, the shunt was patent and normal liver architecture was preserved. The second patient presented with a fulminant course culminating in acute hepatorenal syndrome. There was marked hypofibrinogenimia and extreme elevation of SGOT. The normal thrombocyte count as well as the normal level fibrin degradation products suggested reduced fibrinogen production by the liver as the cause of hypofibrinogenimia. Emergency portacaval shunt was followed by improvement in liver status but the patient died of respiratory complications on the 8th postoperative day. The third patient was on conservative treatment only and remained well for 20 months at which time progression of the hepatic vein thrombosis was diagnosed and immediate operative decompression of the liver was advised. The patient refused surgery for some time and died following an emergency portacaval shunt operation. In this patient excessive renal tubular reabsorption of sodium at a distal site was documented. This may explain the generalized edema formation in patients with Budd-Chiari syndrome even without inferior vena cava obstruction.

1333 HYPERTRIGLYCERIDAEMIA SECONDARY TO LIVER DISEASE. *(E.)* Muller, P.; Fellin, R.; Lambrecht, J.; Agostini, B.; Wieland, H.; Rost, W.; Seidel, D. (Univ. Med. Clin., Heidelberg, Germany). *Eur. J. Clin. Invest.* 4:419-428, 1974.

An abnormally large (300-700 Å), triglyceride rich, low density lipoprotein (density, 1.019-1.063 g/ml) was identified, isolated, and characterized from the plasma of 15 of 22 patients with hypertriglyceridemia secondary to liver disease and designated β_2-lipoprotein. On the basis of its immunoelectrophoretic pattern, β_2-lipoprotein could not be identified in liver disease patients without hypertriglyceridemia. Immunoelectrophoretic analysis of the patients' low density lipoprotein serum fraction in 1% agar gel, indicated 3 different lipoprotein compounds in contrast to 1 lipoprotein in the serum low density lipoprotein fraction of healthy controls. These 3 lipoproteins were separated and isolated using a combination of ultracentrifugation, cold ethanol fractionation, and affinity chromatography with specific antibodies and were found to be 1) the lipoprotein characteristic of cholestasis (lipoprotein-X), 2) normal β-lipoprotein, and 3) β_2-lipoprotein. The immunological properties of isolated β_2-lipoprotein were

tested by double immunodiffusion and immunoelectrophoresis; it never reacted with antibodies to human albumin or human globulins and no constant immunoreaction was obtained with anti-apo A serum. Each of 5 different preparations, however, showed a precipitin line in identity with antibodies to apo-B and apo-C in both double immunodiffusion and immunoelectrophoresis, indicating that these 2 apo-lipoproteins take part in the structure of this plasma lipoprotein. This possibility is supported by the results of a dietary experiment in which β_2-lipoprotein concentration decreased in the plasma of patients on a low fat diet. It is suggested that β_2-lipoprotein is a degradation product or intermediate particle of chylomicron metabolism and that one significant mechanism responsible for its accumulation in this disease is the markedly reduced hepatic lipase activity demonstrated in these patients.

1334 LIVER DISEASE IN PATIENTS WITH JOINT SYMPTOMS. *(E.)* Hilton, A. M.; Boyes, B. E.; Smith, P. J.; Sharp, J.; Dymock, I. W. (Univ. Hosp. South Manchester, England). *Ann. Rheum. Dis.* 33(6): 540-547, 1974.

The results of detailed hepatic investigations are reported for 30 patients who presented with joint symptoms over a 2-yr period at a rheumatology clinic, and who were found on routine clinical examination or biochemical screening to have hepatomegaly and/or abnormal routine liver function tests. In 20 patients, a specific hepatic disorder was found and a varied spectrum of liver disease revealed. The diagnoses included alcoholic liver disease (3 patients), drug-induced liver damage (3), primary biliary cirrhosis (3), chronic persistent hepatitis (3), chronic active hepatitis (2), polyarteritis nodosa, sarcoidosis, cardiac cirrhosis, and severe fatty change (1 each). In 10 patients no definite diagnosis was reached to explain the hepatic abnormalities. In the 3 patients with drug-induced liver damage, the disturbances of liver function were attributable to drug therapy (indomethacin or azathioprine) which resolved when the drugs were withdrawn, illustrating the importance of drug toxicity as a cause of liver abnormalities in patients with rheumatological disorders. It is suggested that detailed hepatic investigations are justified in patients with rheumatic disorders who have hepatomegaly and/or abnormal routine liver function tests; such investigations may reveal a variety of subclinical liver diseases, some of which may be treatable or reversible.

1335 HEPATIC AND SPLENIC LYMPHANGIOMATOSIS WITH SKELETAL INVOLVEMENT: REPORT OF A CASE AND REVIEW OF THE LITERATURE. *(E.)* Asch, M. J.; Cohen, A. H.; Moore, T. C. (Harbor Gen. Hosp., Torrance, Calif.). *Surgery* 76(2):334-339, 1974.

A case of lymphangiomatosis with hepatic and splenic calcifications is presented. A laparotomy was performed on a 10-yr-old girl with hepatosplenomegaly and lytic lesions of the 2nd and 4th ribs. An enlarged spleen was removed that contained cysts filled with amorphous eosinophilic debris, cholesterol crystals, calcium, and hemosiderin. The enlarged liver was diffusely cystic for 60% of the parenchyma with normal tissue intervening. The postoperative course was uncomplicated. After 1 yr of follow-up, the patient remains active and well; the liver size is unchanged as per physical examination; scan and liver chemistries remain normal, and skeletal lesions are unchanged. Prognosis is poor for those patients who have liver and/or spleen involvement along with skeletal angiomatosis and in patients with cystic lymphangiomatosis involving nonintegumental extraskeletal structures. Cystic lymphangiomatosis is likely to be progressive; if there is only limited liver involvement, surgical removal is to be considered. With diffuse disease, there may be little to offer, e.g., transplantation.

1336 BILE ACID METABOLISM AND HEPATIC DISEASE FOLLOWING SMALL BOWEL BYPASS FOR OBESITY. *(E.)* Sherr, H. P.; Nair, P. P.; White, J. J.; Banwell, J. G.; Lockwood, D. H. (Johns Hopkins Univ. Sch. Med., Baltimore, Md.). *Am. J. Clin. Nutr.* 27(12): 1369-1379, 1974.

A possible relation of altered bile acid metabolism to liver disease following jejunoileal bypass for morbid obesity was investigated in 5 patients, 2 men and 3 women aged 23-36 yr. In the 4 months after operation, the patients lost from 68 to 107 pounds each. In all patients prior to operation, liver function tests, as well as prothrombin time, fecal fat and carotene were essentially normal. At 4 months postoperatively, there was steatorrhea, diarrhea, abnormal absorption of B_{12} and D-xylose, a fall in serum cholesterol and carotene, impaired liver function, significant hepatic steatosis, and in 1 patient, the appearance of stellate fibrosis. The relative percentages of bile acids in bile were unaltered, but in 3 patients there was a postoperative increase in the ratio of glycine to taurine bile acid conjugates. These latter changes could not be explained by a taurine deficiency, as plasma taurine levels were unchanged. In 4 patients marked elevation in serum bile acids occurred postoperatively, predominantly in cholic acid and chenodeoxycholic acid. In the 1 patient who developed progressive fibrosis, serum cholic acid was increased 192-fold, chenodeoxycholic acid 195-fold and lithocholic acid 14-fold. However, biliary lithocholic acid in this patient was entirely in the water-soluble and presumbably rapidly excretable sulfated form, factors which rule against the pathophysiological role of lithocholic acid in the development of liver disease. Analysis of bile acids in this patient's liver revealed an absence of lithocholic acid and an elevation in chenodeoxycholic acid to levels known to cause hepatic injury. From these studies it appears that altered bile acid metabolism is not the sole factor responsible for all of the hepatic changes following surgery. It is proposed, however, that increased levels of chenodeoxycholic acid in association with protein malnutrition may be responsible for postoperative hepatic fibrosis. It is recommended that periodic liver biopsies be employed in patients such as these to ensure proper follow-up.

1337 CLINICAL APPLICATION OF CYTOCHROME a($+a_3$) ASSAY OF MITOCHONDRIA FROM LIVER SPECIMENS: AN AID IN DETERMINING METABOLIC TOLERANCE OF LIVER REMNANT FOR HEPATIC RESECTION. *(E.)* Ozawa, K.; Yamaoka, Y.; Kitamura, O.; Nambu, H.; Kamiyama, Y.; Takeda, H.; Takasan, H.; Honjo, I. (Fac. Med., Kyoto Univ., Japan). *Ann. Surg.* 180(6):868-876, 1974.

Measurement of cytochrome a($+a_3$) contents in liver mitochondria was made on 52 biopsy specimens of patients with liver tumor. Seventeen patients had primary tumors of the liver. The others had secondary tumors arising from the stomach, 23; colon and rectum, 5; pancreas, 6; and gallbladder, 1. There were 36 men and 16 women, aged 8 months to 72 yr. Of these 52 patients, 12 had undergone liver resection for cancer, including 3 extended right hepatectomies. Patients having higher cytochrome a($+a_3$) contents in mitochondria from remaining liver than those of normal human liver mitochondria adequately survived major liver resections whether or not associated with liver cirrhosis. However, patients with cytochrome a($+a_3$) contents less than 0.5×10^{-10} M/mg protein showed a high rate of postoperative complication (80%) and death (40%) in spite of minor operation. In routinely used liver function tests such as serum albumin, albumin:globulin ratio, SGOT, total bilirubin, prothrombin time, BSP, and thymol turbidity test, there were no significant differences between patients with cytochrome a($+a_3$) contents more than 0.5×10^{-10} M/mg protein and those less than 0.5. These results indicate that routine laboratory studies do not bear much diagnostic value in estimating a marked decrease of mitochondrial cytochrome a($+a_3$) contents. It is suggested that the measurements of cytochrome a($+a_3$) of the remnant liver should be done prior to a contemplated major resection.

1338 EXCHANGE TRANSFUSION IN HEPATIC COMA: FACTORS AFFECTING RESULTS, WITH LONG-TERM FOLLOW-UP DATA. *(E.)* Lewis, J. D.; Hussey, C. V.; Varma, R. R.; Darin, J. C. (Med. Coll. Wisconsin, Milwaukee). *Am. J. Surg.* 129(2):125-129, 1975.

Exchange transfusion as a therapeutic measure in fulminant hepatic failure was studied in 19 patients aged 7-71 of both sexes. Twelve had infectious hepatitis, 2 had Laennac's cirrhosis, 2 had postoperative complications, and 1 each had serum hepatitis, isoniazid hepatitis, and avascularnecrosis. Arteriovenous shunts were used, except in children too small for arterial lines, in which case venovenous exchanges were used. The active rate of exchange averaged 200 cc/min, but the total time involved was 3-4 hr. Each patient's blood volume was determined with a double tag radioisotope technique, using ^{151}Cr and radioiodinated serum albumin. The exchange volume was 150% of the calculated blood volume. Survival over 2 weeks was considered to constitute response to therapy; response rate was 42%. A 4-5 year follow-up on the patients who survived showed no apparent sequelae. Of the 4 who survived, 3 had infectious hepatitis, and 1 Laennac's cirrhosis. Mortality among the others was mainly due to sepsis. Two biochemical changes were observed 6 hr after exchange: rebound of serum bilirubin and a serum ammonia level over 200 μg/100 ml which indicated a grave prognosis. The results do not indicate that exchange transfusion is the answer to therapy of fulminant hepatic failure; the therapy is, however, of prognostic value.

1339 CORTICOSTEROIDS, ANTIMETABOLITES, AND THE LIVER. *(Sp.)* Rull Segura, S. (Fac. Med., Univ. Navarra, Pamplona, Spain). *Rev. Esp. Enferm. Apar. Dig.* 44(4):409-420, 1974.

1340 BLOOD IMMUNOGLOBULIN (IgA, IgG, AND IgM) LEVELS AND LIVER DISEASE. *(It.)* Squadrito, G.; Ceruso, D.; Quartarone, M.; Monacciani, U. (Inst. Med. Symptomatol., Univ. Messina, Italy). *Boll. Soc. Ital. Biol. Sper.* 49(21):1260-1264, 1973.

1341 ALKALINE PHOSPHATASE ISOENZYMES IN LIVER DISEASE. *(Fr.)* Petitclerc, C.; Briere, N. (Hosp. Cent., Univ. Sherbrooke, Canada). *Union Med. Can.* 103(12):2132-2136, 1974.

1342 ALPHA-FETAPROTEIN IN CLINICAL PATHOLOGY. A REVIEW. *(Fr.)* Belanger, L. (Fac. Med., Laval Univ., Quebec, Canada). *Union Med. Can.* 103(12):2123-2131, 1974.

1343 HETEROTOPIC TRANSPLANTATION OF THE LIVER. *(Fr.)* Geaudoin, J. G.; Maclean, L. D. (Dep. Surg., McGill Univ., Montreal, Canada). *Union Med. Can.* 103(12):2110-2115, 1974.

1344 SERUM MIGRATION INHIBITORY ACTIVITY IN PATIENTS WITH POSTTRANSPLANTATION HEPATIC DYSFUNCTION. *(E.)* Torisu, M.; Yoshida, T.; Cohen, S. (Univ. Connecticut Health Cent., Farmington). *Clin. Immunol. Immunopathol.* 3(3):369-376, 1975.

1345 MAJOR HEPATIC RESECTIONS IN CHILDREN. *(E.)* Stone, H. H. (Emory Univ. Sch. Med., Atlanta, Ga.). *J. Pediatr. Surg.* 10(1):127-134, 1975.

1346 INFLUENCE OF LIVER RESECTION ON SOME CHARACTERISTICS OF LIVER FUNCTION. *(Rus.)* Zhuravlev, V. A.; Voronchikhina, L. D.; Vasilev, P. N.; Korepanov, V. I.; Shestakova, E. V. (Kirovsk Res. Inst. Blood Transfus., USSR). *Sov. Med.* (12):85-88, 1973.

1347 THERAPY OF ACUTE LIVER INSUFFICIENCY. *(Ger.)* Meyer zum Buschenfelde, K. H. (2nd Med. Clin., Univ. Mainz, Germany). *Langenbecks Arch. Chir.* 337:235-243, 1974.

1348 CONJUGATION OF ^{14}C-CHOLIC ACID IN PATIENTS WITH FULMINANT HEPATIC FAILURE. *(E.)* Horak, W.; Murray-Lyon, I. M.; Waldram, R.; Williams, R. (King's Coll. Hosp., London, England). *Digestion* 10(4/5):356-357, 1974.

1349 CALCIFIED MIXED MALIGNANT TUMOR OF THE LIVER. *(E.)* Ludwig, J.; Grier, M. W.; Hoffman, II, H. N.; McGill, D. B. (Mayo Clin., Rochester, Minn.). *Arch. Pathol.* 99(3):162-166, 1975.

1350 ASSOCIATION OF HEPATOMA AND AUSTRALIA ANTIGEN: ONE CASE STUDIED BY ELECTRON MICROSCOPY AND IMMUNOFLUORESCENCE. *(Fr.)* Bodin, F.; Scotto, J.; Homberg, J. C.; Lichtenstein, H.; Roudier, M.; Huguet, C. (St. Antoine Hosp., Paris, France). *Nouv. Presse Med.* 3(38/40):2487-2489, 1974.

1351 DIARRHEA SYNDROME IN HEPATIC ADENOMA. *(E.)* Minow, R. A.; Tompkins, R. K.; Gitnick, G. L. (Univ. California Los Angeles Cent. Health Sci.). *Am. J. Dig. Dis.* 20(2):182-186, 1975.

1352 HAEMOPERITONEUM COMPLICATING THOROTRAST-INDUCED HAEMANGIOENDOTHELIOMA OF THE LIVER. *(E.)* Campbell, I. W.; Webb, J. N. (R. Infirm., Edinburgh, Scotland). *J. R. Coll. Surg. Edinb.* 19(4): 233-237, 1974.

1353 PROTRACTED COURSE OF PRIMARY CANCER OF THE LIVER. *(Rus.)* Piatak, O. A.; Simbiriakova, K. I.; Polischuk, M. L. (A. M. Gorky Med. Inst., Donetsk, USSR). *Ter. Arkh.* 46:91-93, 1974.

1354 PRIMARY TUMORS AND MALFORMATIVE CYSTS OF THE LIVER IN ADULTS. *(Fr.)* Lanitis, G.; Heredia, A.; Ryncki, P. V. (Inst. Pathol., Univ. Lausanne, Switzerland). *Helv. Chir. Acta* 41(5/6): 715-723, 1974.

1355 COMBINED CHEMOTHERAPY AND SURGERY IN THE TREATMENT OF PRIMARY AND SECONDARY LIVER TUMORS. *(Fr.)* Morgenthaler, F. R.; Pettavel, J. (no affil.). *Helv. Chir. Acta* 41(5/6):725-731, 1974.

1356 PARTIAL HEPATECTOMY FOR HEPATIC METASTASES. *(Ger.)* Wanger, F. (Inselspital, Bern, Switzerland). *Helv. Chir. Acta* 41(5/6):733-738, 1974.

1357 DISSEMINATED INTRAVASCULAR COAGULATION AND FATAL THROMBOSIS IN A PATIENT WITH CARCINOMA OF THE BILIARY TRACT. *(Ger.)* Kunz, F.; Amor, H.; Hortnagl, H.; Wesier, G.; Holzknect, F.; Braunsteiner, H. (Med. Clin., Univ. Innsbruck, Austria). *Dtsch. Med. Wochenschr.* 99(52):2643, 1974.

1358 LIPIDS OF CULTURED HEPATOMA CELLS: IV. EFFECT OF SERUM AND LIPID UPON CELLULAR AND MEDIA NEUTRAL LIPIDS. *(E.)* Wood, R.; Falch, J. (Univ. Missouri Sch. Med., Columbia). *Lipids* 9(12):979-986, 1974.

1359 LIPIDS OF CULTURED HEPATOMA CELLS: V. DISTRIBUTION OF ISOMERIC MONOENE FATTY ACIDS IN INDIVIDUAL LIPID CLASSES. *(E.)* Wood, R.; Falch, J.; Wiegand, R. D. (Univ. Missouri Sch. Med., Columbia). *Lipids* 9(12):987-992, 1974.

1360 SURGICAL TREATMENT OF TRAUMATIC RUPTURE OF THE LIVER. *(Ger.)* Bekier, J.; Gonzenbach, H. (Uster Reg. Hosp., Switzerland). *Helv. Chir. Acta* 41(4):505-512, 1974.

1361 SURGERY FOR COARCTATION OF THE INFERIOR VENA CAVA ASSOCIATED WITH BUDD-CHIARI SYNDROME. *(Rus.)* Pokrovskii, A. V.; Apsatarov, E. A.; Kiiashko, V. A.; Krestinskaia, N. N. (A. N. Bakulev Inst. Cardiovasc. Surg., Moscow, USSR). *Vestn. Khir.* 113(9):18-23, 1974.

1362 ACCUMULATION OF OXYGENATED STERYL ESTERS IN WOLMAN'S DISEASE. *(E.)* Assmann, G.; Fredrickson, D. S.; Sloan, H. R.; Fales, H. M.; Highet, R. J. (Natl. Heart Lung Inst., Bethesda, Md.). *J. Lipid Res.* 16(1):28-38, 1975.

1363 DEFECTIVE BILE ACID SYNTHESIS IN CEREBROTENDINOUS XANTHOMASTOSIS. *(E.)* Salen, G.; Shefer, S.; Tint, G. S.; Setaguchi, T.; Mosbach, E. H. (VA Hosp., East Orange, N.J.). *Gastroenterology* 67(4):825, 1974.

1364 INTRAHEPATIC CHOLESTASIS OF PREGNANCY. *(Ger.)* Krejs, G. J.; Blum, A. L. (Triemli Munic. Hosp., Zurich, Switzerland). *Munch. Med. Wochenschr.* 116(51/52):2237-2242, 1974.

1365 INTRAHEPATIC CHOLESTATIS. *(Ger.)* Beran, R. (Prov. Hosp., Graz, Austria). *Wien. Med. Wochenschr.* (suppl. 22):3-16, 1974.

1366 RHEOGRAPHY OF THE LIVER IN CHILDREN WITH LEUKEMIA. *(Rus.)* Podgalo, D. A. (Inst. Pediatr., Moscow, USSR). *Pediatriia* 8:20-23, 1974.

1367 SOME ENZYMOLOGICAL CRITERIA IN SERA FROM CHILDREN WITH VARIOUS LIVER DISEASES AND ACUTE LEUKEMIA. *(Rus.)* Vaganov, P. D.; Zubikova, I. I. (N. I. Pirogov 2nd Moscow Med. Inst., USSR). *Pediatriia* (8):23-26, 1974.

See also, 1002, 1029, 1143, 1144, 1145, 1149, 1179, 1180, 1181, 1182, 1183, 1185, 1186, 1187, 1188, 1189, 1190, 1191, 1192, 1193, 1194, 1195, 1200, 1201, 1202, 1402, 1429, 1435, 1438, 1439, 1468, 1486, 1501, 1537, 1585, 1586, 1591, 1592, 1596.

LIVER AND BILIARY TRACT

Hyperbilirubinemic States

1368 α_1-FETOPROTEIN IN SEVERE NEONATAL JAUNDICE. *(It.)* Gravina, E.; Gravina-Sanvitale, G. (Cologna Veneto Gen. Reg. Hosp., Verona, Italy). *Fegato* 19(2):205-210, 1973.

1369 DIAGNOSIS OF JAUNDICE RESULTING FROM A CONGENITAL ENZYME DEFICIENCY OF LIVER CELLS. *(Fr.)* Magnenat, P.; Okolicsanyi, L. (Dep. Med., Univ. Lausanne, Switzerland). *Fegato* 19(2): 343-351, 1973.

1370 NEONATAL JAUNDICE AND THE DIFFERENTIAL DIAGNOSIS OF NEONATAL HEPATOSPLENOMEGALY. *(Ger.)* Bachmann, K. D. (Pediatr. Clin., Westphalian Wilhelms Univ., Munster, Germany). *Geburtshilfe. Frauenheilkd.* 34(8):597-606, 1974.

1371 CHRONIC HYPERBILIRUBINEMIA IN CHILDREN AND ADULTS DUE TO A CONGENITAL GLUCURONYL TRANSFERASE DEFICIENCY. *(It.)* Gentilini, P.; Ciampolini, M.; Bartolozzi, G.; Surrenti, C. (Inst. Clin. Gen. Med., Med. Ther., Univ. Florence, Italy). *Fegato* 19(2):443-456, 1973.

1372 CONGENITAL IMPAIRMENT OF THE MECHANISM BINDING BILIRUBIN TO GLUCURONIC ACID (CRIGLER-NAJJAR AND RELATED SYNDROMES). *(It.)* Teodori, U. (Inst. Med. Clin., Univ. Florence, Italy). *Fegato* 19(2):435-442, 1973.

1373 GILBERT'S SYNDROME AND VITAMIN K-DEPENDENT CLOTTING FACTORS. *(It.)* Ninno, E. D.; Ideo, G.; Mannucci, P. M. (Inst. Spec. Med. Pathol., Milan, Italy). *Fegato* 19(2):429-434, 1973.

1374 CHANGES IN BILIRUBIN CLEARANCE FOLLOWING PHENOBARBITAL TREATMENT IN PATIENTS WITH GILBERT'S SYNDROME AND OTHER FORMS OF HYPERBILIRUBINEMIA. *(It.)* Surrenti, C.; Chirantini, E.; Salvadori, G.; Casini, M. C.; Buzzelli, G.; Gentilini, P. (Inst. Clin. Gen. Med., Univ. Florence, Italy). *Fegato* 19(2):401-409, 1973.

1375 PHARMACOLOGICAL EFFECTS ON AND BILIRUBIN METABOLISM IN THE LIVER: BILIARY EXCRETION AND MICROSOMAL ENZYME INDUCTION. *(It.)* Messini, M.; Grossi, F.; Messina, B. (Sch. Spec. Liver Metab. Dis., Rome, Italy). *Fegato* 19(2):273-291, 1973.

1376 SYNTHESIS AND PLASMA TRANSPORT OF BILIRUBIN. *(It.)* Perona, G. (Inst. Med. Clin., Univ. Padua, Verona, Italy). *Fegato* 19(2):93-107, 1973.

1377 DRUGS AND CHEMICALS WHICH INTERFERE WITH BILIRUBIN METABOLISM. *(It.)* Cugurra, F. (Inst. Pharmacol. Pharmacog., Univ. Genoa, Italy). *Fegato* 19(2):211-223, 1973.

See also, 1088, 1455.

LIVER AND BILIARY TRACT

Nonviral Hepatitis and Hepatic Injury

1378 THE LIVER IN BOUTONNEUSE FEVER. *(E.)* Guardia, J.; Martinez-Vazquez, J. M.; Moragas, A.; Rey, C.; Vilaseca, J.; Tornos, J.; Beltran, M.; Bacardi, R. (Munic. Soc. Welfare Hosp., Barcelona, Spain). *Gut* 15(7):549-551, 1974.

Hepatic lesions were studied in 13 cases of boutonneuse fever (Mediterranean exanthematous fever). SGOT values were elevated in 8 patients, SGPT values in 10 patients, alkaline phosphatases in 7 patients, and conjugated bilirubin in 3 patients. Of 5 patients studied histologically, 3 had lesions of a granulomatous type, 1 fatty degeneration with alcoholism, and the 5th had a normal liver. Two of the three patients with granulomatous lesions showed a moderate increase in alkaline phosphatases, but transaminase values were nearly normal. The hepatic changes had no clinical repercussions. Although no assertions can be made concerning the mechanism that causes the lesions, boutonneuse fever should be included among infectious conditions that can produce granulomas within the liver.

1379 VINYL-CHLORIDE-INDUCED LIVER DISEASE: FROM IDIOPATHIC PORTAL HYPERTENSION (BANTI'S SYNDROME) TO ANGIOSARCOMAS. *(E.)* Thomas, L. B.; Popper, H.; Berk, P. D.; Selikoff, I.; Falk, H. (Natl. Inst. Health, Bethesda, Md.). *N. Engl. J. Med.* 292(1):17-22, 1975.

Histologic examination was made of liver tissue (8 autopsy and 18 biopsy specimens) and 5 spleens from 20 workers who had industrial exposure to vinyl chloride for periods usually exceeding 2 years and extending up to 18 years. Tissue samples were taken from 15 patients with angiosarcoma and 5 with hepatic fibrosis but without angiosarcoma. Three basic patterns were noted in the different parts of the angiosarcomas: the sinusoidal pattern, the papil-

lary pattern, and the cavernous pattern. In addition, 1/4 of the angiosarcomas had more solid nodules of anaplastic sarcoma. In 3 patients, organs other than the liver were involved by angiosarcoma: the duodenum in 1, the lung in another, and the lung, heart, kidney, and lymph nodes in the third. In addition, a peculiar pattern of progressive portal-tract, inconspicuous intralobular and conspicuous capsular fibrosis was observed in the 5 workers without angiosarcoma, in all 7 patients with angiosarcoma from whom tumor-free portions of the liver were available, and in 2 tumor-free biopsies from patients subsequently found to have angiosarcoma. The fibrosis was accompanied by splenomegaly. Hypertrophy and hyperplasia of both hepatocytes and hepatic and splenic mesenchymal cells were also seen. The histologic similarity to chronic inorganic arsenical poisoning, in which angiosarcomas also occur, and to idiopathic portal hypertension (Banti's syndrome) suggests that the latter syndrome at times results from unknown toxic, possibly environmental, chemicals. This study demonstrates a stimulating effect by vinyl chloride or its metabolites on several types of hepatic and splenic cells and suggests a transition from the fibrotic stage to angiosarcoma.

1380 LIVER DISEASE CAUSED BY PROPYLTHIOURACIL. *(E.)* Fedotin, M. S.; Lefer, L. G. (Duke Univ. Med. Cent., Durham, N.C.). *Arch. Intern. Med.* 135(2):319-321, 1975.

A 56 yr old male presented with a 2-yr history of weakness, 50 lb weight loss, insomnia, hyperdefecation, heat intolerance, and severe tremors. He had taken diazepam (5 mg as needed) for several years. Two and a half months prior to admission hyperthyroidism was diagnosed. Alkaline phosphatase value was raised to 5 Bodansky U, and bilirubin value was 1.4 mg/100 ml. He was given propylthiouracil (50 mg, twice/day). During further evaluation, the propylthiouracil was increased to 100 mg, 3 times/day and the diazepam to 5 mg, 4 times/day. At this time the bilirubin value was 2.1 mg/100 ml, alkaline phosphatase level was 395 IU, SGOT 384, SGPT 238 U. After 2 days the bilirubin was 2.0 mg/100 ml, SGOT 596 U, alkaline phosphatase 290 IU. Propylthiouracil therapy was discontinued and a percutaneous liver biopsy was obtained. On the 4th day after discontinuing propylthiouracil therapy the bilirubin level was 0.9 mg/ml, alkaline phosphatase 220 IU, SGOT 89 U, all of which continued to decrease. The histologic and electron microscopic examination of the biopsy material revealed characteristic findings of chronic active (aggressive) hepatitis. The marked increase in liver function test abnormalities, their rapid return to normal, and the close temporal relationship of these changes to manipulations of propylthiouracil therapy suggests that propylthiouracil sensitivity was the likely cause of this patient's liver disease. The continued use of diazepam while the liver function was improving removes this drug from suspicion. It is suggested that propylthiouracil be added to the list of drugs which must be considered in the evaluation of chronic liver disease.

1381 HEPATIC PHARMACOLOGY. *(Fr.)* Arlaud, J. (no affil.). *Marseille Med.* 110(10):763-770, 1973.

1382 LIVER DISEASE AND DRUGS. *(Sp.)* Gonzalez Macias, J.; de Castro del Pozo, S. (Fac. Med., Salamanca, Spain). *Rev. Esp. Enferm. Apar. Dig.* 44(2):175-186, 1974.

1383 LIVER INJURY IN JUVENILE DRUG ADDICTS. *(Ger.)* Brunner, H.; Kroiss, A.; Grabner, G.; Ranefeld, J. (Dep. Gastroenterol., Univ. Vienna, Austria). *Acta Med. Austriaca* 1(2):48-54, 1974.

1384 THE COURSE OF TOXIC HEPATITIS CAUSED BY LONG EXPOSURE TO HEXACHLOROCYCLOHEXANE (HCH) PREPARATIONS. *(Rus.)* Sasinovich, L. M.; Alekhina, S. M.; Odintsova, I. L. (All-Union Sci. Res. Inst. Hyg. Toxicol. Pestic., Polym., Plast., Kiev, USSR). *Vrach. Delo* (10):133-135, 1974.

1385 AN ULTRASTRUCTURAL STUDY OF TETRACYCLINE HEPATOTOXICITY IN MAN. *(E.)* Pittman, F. E.; Ainsworth, S. K.; Hennigar, G. R.; Pittman, J. C. (Med. Univ. South Carolina, Charleston). *Gastroenterology* 67(4):821, 1974.

1386 DRUG-INDUCED LIVER DAMAGE, WITH SPECIAL REFERENCE TO CHANGES AND FUNCTIONAL DISORDERS IN LIVER CELLS AFTER ORAL ADMINISTRATION OF HORMONAL CONTRACEPTIVE DRUGS. *(Pol.)* Raczynska, A. (M. Kopernik Hosp., Torun, Poland). *Patol. Pol.* 25(1):91-100, 1974.

1387 SOME "VIRAL" HEPATITIS IS CAUSED BY RICKETTSIA OR SIMILAR AGENTS. ITS CAUSE SHOULD BE DIAGNOSED SINCE IT IS SENSITIVE TO LARGE ENOUGH DOSES OF BROAD-SPECTRUM ANTIBIOTICS. *(Fr.)* Giroud, P.; Jadin, J. (Pasteur Inst., Paris, France). *Bull. Acad. Natl. Med. (Paris)* 158(1):87-91, 1974.

1388 LIVER DISEASE CAUSED BY CHICKENPOX VIRUS IN CHILDREN WITH LEUKEMIA. *(Rus.)* Talalaev, A. G. (N. I. Pirogov 2nd Moscow Med. Inst., USSR). *Pediatriia* (8):26-31, 1974.

1389 TWO CASES OF ISOLATED TUBERCULOSIS OF THE LIVER. *(Pol.)* Kawecki, J.; Nowoczyn, J. (PCK Nav. Hosp., Gdynia, Poland). *Gruzlica* 41(10):1083-1088, 1973.

1390 TUBERCULOSIS OF THE LIVER. *(Sp.)* Garcia Ocana, A.; Larranaga Barrera, E.; Candia Sierra, A.; Lopez-Yarto, J. (Gran Hosp. Estado, Madrid, Spain). *Rev. Esp. Enferm. Apar. Dig.* 44(4):387-400, 1974.

See also, 0923, 1029, 1348, 1377, 1411, 1444.

1391 ACUTE VIRAL HEPATITIS: A SURVEY OF 500 PATIENTS. CLINICAL, BIOCHEMICAL, IMMUNOLOGICAL, AND MORPHOLOGICAL FEATURES AT TIME OF DIAGNOSIS. *(E.)* Petersen, P.; Christoffersen, P.; Elling, P.; Juhl, E.; Dietrichson, O.; Faber, V.; Iversen, K.; Nielsen, J. O.; Poulsen, H. (Blegdam Hosp., Copenhagen, Denmark). *Scand. J. Gastroenterol.* 9(7):607-613, 1974.

Data were collected from February, 1969 to June, 1972 on 500 patients (11-85 yr old; 33.6% female, 66.4% male) with biopsy-verified acute viral hepatitis. Histological diagnosis of acute viral hepatitis was made when the following changes were present: focal liver cell necrosis, variation of cells and nuclei, Kupffer cell proliferation, acidophilic bodies, and portal inflammation. Nearly half of the 500 patients had a history of drug abuse. Fifteen percent had visited zones where hepatitis is endemic within 18 weeks prior to onset of symptoms. Blood samples had been drawn in 17% of the patients within 3 months of onset and 1.6% of the patients had received blood transfusion within 6 months prior to onset of symptoms. No source of infection could be determined in about 28% of the cases. In 87% of the patients jaundice was present before biopsy. The remaining 13% had anicteric hepatitis. The liver was enlarged in 27% of the patients, while splenomegalia was found in only 1 case. A variety of extrahepatic disorders were found: 9 patients had collagen disease, 8 had endocrine disease, and 19 had had previous allergic manifestations. The biochemical and immunological features of the 500 patients are tabulated as are the histological features. It is suggested that these results may represent a real profile of acute viral hepatitis in Copenhagen because the 500 cases were unselected and comprise 1/4 of all patients reported to the Ministry of Health during the same period from the same area.

1392 VIRAL HEPATITIS. *(E.)* Purcell, R. H. (Natl. Inst. Allergy Infect. Dis., Bethesda, Md.). *Prev. Med.* 3(4):529-534, 1974.

The discovery of hepatitis B and hepatitis A antigens, and the epidemiology, laboratory study, and prospects for control of both hepatitis A and B are reviewed. At least 4 subtypes of hepatitis B surface antigen have been identified, but it is not clear whether these are reflections of different serotypes of hepatitis B virus or whether infection with 1 subtype stimulates immunity to all the subtypes. The application of sensitive assays for hepatitis B antigen and antibody to epidemiologic studies has revealed that hepatitis B virus infection is widespread throughout many populations and appears to be directly related to age and inversely related to socioeconomic status. Hepatitis B virus has not been unequivocally isolated in tissue or organ culture and serially passaged in such *in vitro* systems, but it has been successfully transmitted to chimpanzees and rhesus monkeys. Three approaches to the control of hepatitis B infection are being studied: 1) interdiction of the spread of hepatitis B virus, 2) passive immunization with hepatitis B immune serum globulin, and 3) active immunization with hepatitis B surface antigen. Hepatitis A antigen, which is morphologically and serologically distinct from hepatitis B antigen, has recently been revealed by immune electron microscopy studies in the stools of patients acutely ill with hepatitis A infection. Preliminary epidemiologic studies suggest that more than 2 hepatitis viruses exist, but proof is lacking for a "hepatitis type C". Hepatitis A virus has not been successfully isolated and cultivated *in vitro* but it has been successfully transmitted to marmosets and chimpanzees. It may be possible to improve the protective effect of immune serum globulin against hepatitis A by selecting lots of γ-globulin rich in antibody to hepatitis A antigen; studies to confirm this have not been carried out.

1393 ON THE TRAIL OF EPIDEMIC HEPATITIS. *(E.)* Zuckerman, A. J. (no affil.). *Nature* 252(5480):193-194, 1974.

Virus-like particles, 27 nm in diameter, were demonstrated by immune electron microscopy in fecal extracts from 2 of 4 volunteers infected by injection or p.o. with the MS-1 strain of hepatitis A virus. The particles were heavily coated with antibody and were aggregated by convalescent serum, by serum from 6 volunteers previously infected with hepatitis A, and by serum from patients with naturally occurring hepatitis A. In another study, fecal extracts obtained 33 days after infection with the MS-1 strain of hepatitis A contained virus-like particles measuring 22, 27, and 30 nm in diameter. This material induced hepatitis in 2 of 6 marmosets, suggesting that hepatitis A virus is indeed present.

1394 THE ROLE OF CIRCULATING HEPATITIS B ANTIGEN/ANTIBODY IMMUNE COMPLEXES IN THE PATHOGENESIS OF VASCULAR AND HEPATIC MANIFESTATIONS IN POLYARTERITIS NODOSA. *(E.)* Trepo, C. G.; Zuckerman, A. J.; Bird, R. C.; Prince, A. M. (New York Blood Cent., N.Y.). *J. Clin. Pathol.* 27(11):863-868, 1974.

To investigate the role of hepatitis B antigen (HB_sAg) and specific immune complexes in polyarteritis, sera from 55 histologically confirmed cases were tested for the presence of hepatitis B antigen-associated particles and hepatitis B-antibody (HB_sAb) by solid phase radioimmunoassay, electron microscopy, and passive hemagglutination. Results of these findings were correlated with the clinical course of the disease. HB_sAg was detected in 30 of 55 patients (54.5%) and HB_sAb in 13 of 45 patients (28%). Subtyping in 10 patients revealed that 11 were Y and 9 D. Thirty-seven cases (69%) demonstrated either HB_sAg or HB_sAb and 5 of 45 (11%) had both. Electron microscopic examination showed 20 nm spherical and tubular particles in sera of 20 of 27 patients with 42 nm particles in 11 cases and clumped particles in 12 (60%). No correlation was found between detection of immune complexes and liver disease whereas the presence of co-existing HB_sAg and HB_sAb or aggregated particles was restrict-

ed to cases of active vasculitis. Seroconversion or the presence of HB_sAb alone was associated with improved prognosis. It is suggested that circulating hepatitis B antigen antibody complexes may be responsible for vasculitis or polyarteritis; they do not appear to be pathogenic for the liver.

1395 THE COVERT LIVER DISEASE OF DRUG ADDICTS. *(E.)* Ireton, H. J. C.; Gust, I. D.; Moon, W. J.; Lehmann, N.; Stening, G. F.; Smallwood, R. A. (Austin Hosp., Heidelberg, Australia). *Aust. NZ J. Med.* 4(5):444-449, 1974.

Twenty narcotic addicts (13 male, 7 female, 18-27 yr old) who had been using drugs i.v. for at least 2 yr were examined for the presence of liver disease in an attempt to clarify the etiology and significance of the liver lesion found in these patients. Three narcotic addicts who had been taking drugs orally for more than 25 yr were also studied. The following investigations were performed: chest x-ray, hemoglobin and blood film, WBC, liver function tests, serum protein electrophoresis, immunoelectrophoresis, anti-nuclear factor, anti-smooth muscle and anti-mitochondrial antibodies. Hepatitis-B antigen and antibody were evaluated and liver biopsies processed by standard histological techniques. Eleven (55%) of the patients had abnormal liver function tests, but the abnormalities were limited to minor elevations of SGOT (41-98 Karmen U/ml) and alkaline phosphatase (17-32 King Armstrong U/100 ml). BSP retention was increased in 7 patients. Abnormal liver biopsies were seen in 19 (95%) patients, 14 of which showed the changes of chronic persistent hepatitis, and the remaining 5 showed in addition greatly expanded portal tracts with dense lymphoid aggregates and poorly formed histiocytic granulomas. Foreign material was identified in 5 biopsies. No patient had hepatitis B antigen in the serum, but 4 had detectable levels of hepatitis-B antibody. No liver abnormality was seen in the 3 patients addicted to oral narcotics. It seems unlikely, therefore, that the lesion results from a direct hepatotoxic action of narcotics. It is concluded that the liver lesion is associated with the repeated i.v. injection of contaminated (either chemical or infective) drug preparations.

1396 ROLE OF SALIVA, URINE AND FECES IN THE TRANSMISSION OF TYPE B HEPATITIS. *(E.)* Villarejos, V. M.; Visona, K. A.; Gutierrez, A.; Rodriguez, A. (Louisiana State Univ. Int. Cent. Med. Res. Train., San Jose, Costa Rica). *N. Engl. J. Med.* 291(26):1375-1378, 1974.

The feces, urine and saliva of chronic carriers of hepatitis B antigen (HB Ag) and of patients with acute Type B hepatitis were examined by radioimmunoassay and counterelectrophoresis to assess their importance as vehicles of transmission of the infection. Repeated samples of serum, saliva, urine, and feces from 93 chronic carriers detected in 2 counties of Costa Rica where both Type A and Type B hepatitis occur endemically, and from 120 household contacts, were tested for HB Ag. The antigen was present in none of 120 fecal extracts, in three of 130 urine specimens, and was intermittently present in the saliva in 86% of antigenemic carriers. Serum and saliva from 41 acute hepatitis B patients were examined at weekly intervals during the course of the disease. HB Ag was detected in the saliva of 76% of patients with acute hepatitis during the first 3 weeks after clinical onset; 35% of sneeze samples from patients were antigen positive. Antibody, detected by passive hemagglutination, was found in saliva early during clinical disease, as well as during convalescence. Saliva is probably the main vehicle of infection in non-parenterally acquired Type B hepatitis. Transmission may be either airborne, through large droplets expelled by sneezing and coughing, or directly from mouth-to-mouth by kissing or by exchange of chewed objects among children.

1397 EPIDEMIC VIRAL HEPATITIS, TYPE B, IN HOSPITAL PERSONNEL. *(E.)* Williams, S. V.; Huff, J. C.; Feinglass, E. J.; Gregg, M. B.; Hatch, M. H.; Matsen, J. M. (Cent. Dis. Control, Atlanta, Ga.). *Am. J. Med.* 57(6):904-911, 1974.

An investigation of hepatitis in personnel at a university hospital revealed 79 cases of viral hepatitis between January, 1968 and August, 1972 which were apparently not part of a general outbreak in the community. There were approximately 2,700 full-time employees and an additional 500-1000 persons who worked on a part-time basis in the hospital. Two surveys were performed during the investigation. Blood specimens received in each laboratory area over a 24-hr period were examined to compute the percentage of blood in each area that came from patients with renal disease. Personnel who worked on the renal ward and in the clinical laboratories donated blood samples which were examined for hepatitis B surface antigen (HB_sAg) by agar gel diffusion and complement fixation and for antibody to HB_sAg (HB_sAb) by agar gel diffusion and passive hemagglutination. Of those personnel tested for HB_sAg while ill, 71% were positive by agar gel diffusion and 15 of 16 tested for HB_sAb after their recovery were positive. Thirty-four cases of viral hepatitis B occurred in staff who worked on the renal ward and hemodialysis unit, 34 in personnel who worked in the clinical laboratories, and the other 11 primarily in nursing personnel throughout the rest of the hospital. The number of cases correlated temporally with the number of HB_sAg-positive patients undergoing hemodialysis in the hospital. Clinical hepatitis attack rates and HB_sAb prevalence rates correlated with exposure to blood from patients with renal disease, including those who received transplants, both in the renal ward and in the clinical laboratories. Transmission appears to be by accidental and inapparent parenteral routes involving infective blood from patients with renal disease who were chronic carriers of HB_sAg.

1398 ACUTE VIRUS HEPATITIS IN HUMANS. *(Ger.)* Eisenburg, J. (1st Med. Clin., Univ. Munich, Germany). *Naturwissenschaften* 61(7):298-307, 1974.

1399 POST-TRANSFUSION HEPATITIS. *(E.)* Reinicke, V. (Bispebjerg Hosp., Copenhagen, Denmark). *Scand. J. Infect. Dis.* 6(3):285-290, 1974.

1400 CONTRIBUTION OF MICROSCOPY TO THE STUDY OF TYPE B VIRAL HEPATITIS. *(Fr.)* Huang, S. N. (Dep. Pathol., McGill Univ., Montreal, Canada). *Union Med. Can.* 103(12):2140-2145, 1974.

1401 VISUAL DETECTION OF HEPATITIS B ANTIGEN. *(E.)* Giaever, I.; Laffin, R. J. (Gen. Electr. Corp. Res. Develop., Schenectady, N.Y.). *Proc. Natl. Acad. Sci. USA* 71(11):4533-4535, 1974.

1402 AUSTRALIA ANTIGEN. HEPATOCYTES WITH "GROUND-GLASS" TYPE CYTOPLASM. A CASE OF HEPATOMA SUPERIMPOSED ON CIRRHOSIS WITH AUSTRALIA ANTIGEN AND "GROUND-GLASS" HEPATOCYTES. *(Fr.)* Pequinot, H.; Abelanet, R.; Deslignieres, S.; Christoforov, B. (Cochin Hosp., Paris, France). *Ann. Med. Interne (Paris)* 125(6/7):567-569, 1974.

1403 GLUTAMATE DEHYDROGENASE IN HEPATOBILIARY DISEASE. CHANGES IN ACUTE HEPATITIS AND OBSTRUCTIVE JAUNDICE. *(Sp.)* Rodríguez Cuartero, A.; Nunez Carril, J.; Lopez Fernandez, A. (San Cecilio Clin. Hosp., Granada, Spain). *Rev. Esp. Enferm. Apar. Dig.* 44(1):35-44, 1974.

1404 CHANGE IN SALIVARY TRACE ELEMENT CONCENTRATIONS IN VARIOUS STAGES OF VIRAL HEPATITIS. *(Rus.)* Sagradian, A. L. (Tbilisi State Inst. Postgrad. Med., USSR). *Soobshch. Akad. Nauk Gruz. SSR* 76(1):189-191, 1974.

1405 AUSTRALIA-ANTIGEN-POSITIVE TESTS IN HOSPITAL PATIENTS. *(Ger.)* Zollikofer, J.; Frey-Wettstein, M. (Limmattal Blood Donor Cent., Schlieren/Zurich, Switzerland). *Helv. Med. Acta* 37(5/6):401-402, 1974.

1406 THE RESEARCH OF BOTH Au ANTIGEN AND ANTI-Au ANTIBODIES AS A DEDUCTIVE TEST FOR HEPATOCELLULAR CHANGES IN CLINICALLY HEALTHY SUBJECTS. *(It.)* Nobili, G.; Adorisio, E.; Zardi, O. (Aldo di Loreto Inst. Forensic Med., Rome, Italy). *Riv. Med. Aeronaut. Spaz.* 36(3/4):165-175, 1973.

1407 HEPATITIS B VIRUS: EPIDEMIOLOGICAL ASPECTS AND IMMUNOLOGICAL BASIS FOR THE PATHOLOGICAL PHYSIOLOGY. *(Fr.)* Richer, G. (Dep. Microbiol. Immunol., Univ. Montreal, Canada). *Union Med. Can.* 103(12):2116-2122, 1974.

1408 ASPECTS OF HEPATITIS EPIDEMIOLOGY IN SWITZERLAND. *(Ger.)* Gassner, M.; Grob, P. J. (Dep. Intern. Med., Univ. Zurich, Switzerland). *Helv. Med. Acta* 37(5/6):399-400, 1974.

1409 HOSPITAL OUTBREAK OF HEPATITIS. III. RELATION BETWEEN THE CLINICAL AND BIOCHEMICAL CHANGES IN SERUM IMMUNOGLOBULINS. *(Sp.)* Borda Celaya, F.; Perez Miranda, M.; Pons Romero, F.; de Prado Marcilla, J. M.; Errasti Goenaga, P.; Sanchez Ibarrola, A. (Fac. Med., Navarra, Spain). *Rev. Esp. Enferm. Apar.* 44(3):219-230, 1974.

1410 AN EPIDEMIOLOGIC STUDY OF HEPATITIS B VIRUS IN BANGKOK, THAILAND. *(E.)* Grossman, R. A.; Benenson, M. W.; Scott, R. M.; Snitbhan, R.; Top, Jr., F. H.; Pantuwatana, S. (Dep. Epidemiol., SEATO, San Francisco, Calif.). *Am. J. Epidemiol.* 101(2):144-159, 1975.

1411 EXPERIENCE IN TREATMENT OF ACUTE HEPATIC FAILURE. *(E.)* Galmarini, D.; Tarenzi, L.; Cantaluppi, G.; Mantovani, A.; Zanadrea, G.; Pesenti, A.; Girardello, D.; Pellegrini, S.; Doglia, M.; Fabiani, M. P.; Vitali, B. (Cent. Exp. Surg., Univ. Milan, Italy). *Digestion* 10(4/5):349, 1974.

1412 THE EFFECT OF REST ON THE COURSE OF VIRAL HEPATITIS. *(Fr.)* Laverdant, C.; Cristau, P.; Essioux, H.; Molinie, C.; Gendron, Y.; Bernard, J.; Kassem, A. (Begin Mil. Hosp., Saint-Mande, France). *Ann. Med. Interne (Paris)* 125(10):759-766, 1974.

1413 PREVENTION OF VIRAL HEPATITIS BY NONSPECIFIC GAMMA GLOBULINS. *(Fr.)* Baylon, H.; Laverdant, C. (Army Dep. Health, Paris, France). *Bull. Acad. Natl. Med. (Paris)* 158(1):32-38, 1974.

1414 DEMONSTRATION BY THE PROSTIGMINE TEST OF PANCREATIC PARTICIPATION IN ACUTE VIRAL HEPATITIS. *(Rum.)* Negomireanu, T.; Gorgan, V. (Clin. Infect. Dis., Cluj, Rumania). *Cluj Med.* 46(1):43-48, 1973.

See also, 1196, 1318, 1341, 1348, 1350, 1422.

1415 PREVENTION OF CHRONIC NEONATAL HEPATITIS B VIRUS INFECTION WITH ANTIBODY TO THE HEPATITIS B SURFACE ANTIGEN. *(E.)* Kohler, P. F.; Dubois, R. S.; Merrill, D. A.; Bowes, W. A. (Univ. Colorado Med. Cent., Denver). *N. Engl. J. Med.* 291(26):1378-1380, 1974.

Four babies born to mothers positive for the hepatitis B surface antigen (HB_sAg) were treated 1-6 days after delivery with antibody to the antigen and remained antigen negative for 5-14 months. In 6 of 7 similar but untreated babies, chronic antigenemia developed within 5-12 weeks of birth and persisted in all up to the age of 36 months. Two of 7 umbilical-cord serum samples tested by radioimmunoassay were positive (treated babies) but not when tested by electroimmunodiffusion or counterelectrophoresis. Repeat radioimmunoassay testing on blood obtained directly from the babies 1 and 6 days after delivery was negative. Therefore, HB_sAg detected in umbilical-cord blood may not by itself indicate either *in utero* infection or even transplacental transfer of HB_sAg. These results are probably a consequence of contamination of the cord blood at the time of collection with HB_sAg-positive maternal blood or secretions. Treatment with specific antibody appears to be effective, as compared with no treatment, in preventing neonatal hepatitis B virus infection. The current prevalence of the surface antigen in mothers at term is 0.83% at 3 Denver hospitals. It is suggested that HB_sAg testing of women at term and HB_sAb treatment of babies at risk has the potential to reduce in part the reservoir of chronic HB_sAg carriers who lack an appropriate history of exposure to hepatitis B virus or acute liver disease.

1416 CIRCULATING THYROXINE IN CHRONIC ACTIVE LIVER DISEASE. *(E.)* Barzilai, D. C.; Soloway, R. D.; Summerskill, W. H. J.; Schoenfield, L. J. (Mayo Clin., Rochester, Minn.). *Isr. J. Med. Sci.* 10(11):1405-1409, 1974.

Serum levels of free and total thyroxine were recorded before and during treatment of 40 patients with chronic active liver disease (9 males 31 females, aged 13-75 yr), none of whom had thyroid disease. The patients were randomly assigned to 1 of 4 treatments: 1) placebo; 2) maintenance with prednisone, 20 mg/day; 3) azathioprine, 100 mg/day; and 4) a combination of prednisone, 10 mg/day and azathioprine, 50 mg/day. Patients were examined prior to treatment and after 2 weeks, and then every 3-12 months for 3 yr. Before treatment, the most common abnormality (29% of cases) was an increased fraction of thyroxine present as free thyroxine; less often, the fraction was decreased. Occasionally, initial levels of free or total thyroxine were low. Abnormal serum thyroxine levels were always improved in patients who responded to therapy, that is in 13 patients who received prednisone or the combination of prednisone and azathioprine and in 9 who received azathioprine or placebo; abnormal serum thyroxine levels remained unchanged in the 18 patients who did not respond. There was no significant correlation between the concentration of the free fraction of thyroxine and levels of SGOT or serum bilirubin. There was, however, a significant relationship between the serum albumin concentration and the free fraction of thyroxine before and during treatment. The values for serum free thyroxine and total thyroxine were not related to any results of the liver function tests. Deficient binding of thyroxine to protein, predominantly involving the pre-albumin fraction and resulting from dysproteinemia associated with active chronic liver disease, is proposed as a basis for the abnormalities reported. The diagnosis of thyroid disease in patients with active liver disease is made difficult by the unreliability of the serum levels of free and total thyroxine as indicators of thyroid function and should be deferred until the patient has responded to treatment or be confirmed by ancillary studies, including the uptake of radioiodine by the thyroid.

1417 LYMPHOCYTE CYTOTOXICITY TO ISOLATED HEPATOCYTES IN CHRONIC ACTIVE HEPATITIS. *(E.)* Thomson, A. D.; Cochrane, M. A. G.; McFarlane, I. G.; Eddleston, A. L. W. F.; Williams, R. (King's Coll. Hosp. Med. Sch., London, England). *Nature* 252(5485): 721-722, 1974.

Lymphocytes from 22 patients with chronic active hepatitis were incubated with rabbit hepatocytes, the concentration of lymphocytes being adjusted to produce a lymphocyte-to-target-cell (hepatocyte) ratio of 400:1. Tests were also carried out in 6 patients with primary biliary cirrhosis, 4 with primary hepatocellular carcinoma, 5 with inactive alcoholic cirrhosis, 1 with idiopathic hemochromatosis and 1 with obstructive jaundice. Significant cytotoxicity was demonstrated in 20 (90%) of the patients with chronic active hepatitis, and was also present in 2 of 6 patients with primary biliary cirrhosis. In the 11 patients with other forms of chronic liver disease, the results were similar to the controls, none showing significant cytotoxicity. The mean cytotoxicity of 4 patients with chronic active hepatitis, when lymphocytes alone were added to the incubation chambers, was 53.6%. The addition of either human or rabbit liver-specific lipoprotein prepared from normal rabbit tissue and human organs, together with the lymphocytes, produced a highly significant reduction in cytotoxicity, 5.3% and 5.5%, resp. These blocking experiments implicate sensitization to human liver-specific lipoprotein in chronic active hepatitis as the important factor in the production of the lymphocyte-mediated hepatocyte injury. Such sensitization might be initiated by structural changes in the membrane lipoprotein following virus or drug-induced liver damage, but the specific mechanism has not been determined.

1418 LEUCOCYTE MIGRATION INHIBITION IN LIVER DISEASE. *(E.)* Vergani, C.; Oldoni, T.; Dioguardi, N. (Inst. Clin. Pathol., Univ. Milan, Italy). *J. Clin. Pathol.* 27(10):772-775, 1974.

The usefulness of the migration inhibitory factor (MIF) test as a diagnostic tool in liver disease was investigated in 88 patients with liver disease and in 24 healthy controls. The patients were divided into those with acute hepatitis (13), chronic persistent hepatitis (11), chronic aggressive hepatitis (24), cryptogenic cirrhosis (23), alcoholic cirrhosis (11), and primary biliary cirrhosis (6). Leukocytes were obtained from peripheral venous blood and placed in capillary tubes in a Teflon ring chamber. About 1 ml of tissue culture medium, with or without antigen, was added to the chamber and the leukocytes allowed to migrate for 20 hr after which the migration index was determined. Fetal liver antigen obtained at necropsy was added to the chamber to give 400 µg protein/ml tissue culture medium. Tests were found to be reproducible with respect to the coefficient of variation and also when they were repeated 3 times on the same patient within 1 week. Migration index in healthy controls varied from 87 to 113%. Significant abnormality in migration index was found only in chronic aggressive hepatitis (inhibition in 16 patients, 67%), in cryptogenic cirrhosis (inhibition in 10, 43%; stimulation in 3, 13%), and in primary biliary cirrhosis (inhibition in 2, stimulation in 2). No correlation was detected between MIF and serum γ-globulin, SGOT, SGPT, total bilirubin, or the presence of hepatitis B antigen. These data provide evidence that cell-mediated immunity is implicated in some forms of chronic liver disease, and indicate that the MIF test is a diagnostically useful tool for some forms of liver disease, especially when liver biopsy is not feasible because of coagulation disorders.

1419 INADEQUATE ANTIBODY RESPONSE TO HBAg OR SUPPRESSOR T-CELL DEFECT IN DEVELOPMENT OF ACTIVE CHRONIC HEPATITIS. *(E.)* Eddleston, A. L. W. F.; Williams, R. (King's Coll. Hosp. Med. Sch., London, England). *Lancet* 2(7896):1543-1545, 1974.

The role of autoimmunity in the pathogenesis of active chronic hepatitis is reviewed. It is theorized that the progressive liver damage of active chronic hepatitis is due to an autoimmune reaction directed against an hepatocyte surface lipoprotein which is initiated in most cases by a hepatitis-B-virus infection. According to this theory, T cells recognizing viral determinants activate B cells responsive to the lipoprotein antigen, and where virus infection persists because of a defect in the antibody response to HB Ag, the autoimmune response is continuously activated. In contrast, in the HB Ag-negative group the virus is eliminated normally and the autoimmune reaction persists because of a defect in the suppressor effect of T cells on B cell activity. A genetic marker of this defect may be the histocompatibility antigen HL-A8 which is found in an increased frequency only in the HB Ag-negative cases. Correcting the basic defects by specific immunotherapy is a possibility for the future.

1420 HUMORAL IMMUNITY IN RELATIVES OF PATIENTS WITH ACTIVE CHRONIC HEPATITIS. *(E.)* Joske, R. A.; Sanderson, C. R. (Dep. Med., Univ. West. Australia, Perth). *Aust. NZ J. Med.* 4(5): 441-443, 1974.

Humoral immunity was investigated in 39 first-degree and 12 second-degree relatives of 14 patients (12 females and 2 males) with active chronic hepatitis. Five spouses were also examined to determine possible environmental factors. Blood was taken from each subject for the following tests: hemoglobin, WBC, erythrocyte sedimentation rate, serum bilirubin, alkaline phosphatase, SGOT, SGPT, serum proteins, quantitative protein electrophoresis, immunoglobulins (A, G, and M), hepatitis B antigen, hepatitis B antibody, thyroglobulin antibody, cryoglobulins, Wassermann reaction, cold agglutinins, and rheumatoid factor. Antinuclear factors, antismooth muscle antibody, antimitochondrial antibody and antiparietal cell antibody were also investigated. None of the relatives had clinical evidence of chronic liver or immunopathic disease. A significant increase was shown in the number of abnormal immunological findings, especially hyperglobulinemia (3 males and 1 female) and the presence of tissue antibodies (13 of 37 tested). There were fewer abnormalities in the 12 second-degree relatives. The 5 spouses were normal to all tests used. These results provide evidence that abnormalities of humoral immunity occur with increased frequency in relatives of patients with active chronic hepatitis compared with control populations. These abnormalities are considered to have a genetic basis because 1) they were present in relatives living apart from the patients but absent in marriage partners; and 2) they were more frequent in first than in second degree relatives. The nature of this genetic factor is not known.

1421 SCINTIGRAPHIC STUDIES IN CHRONIC HEPATITIS. *(E.)* Bozduganov, A.; Maistorski, A.; Janev, P. (I. P. Pavlov Higher Med. Inst., Plovdiv, Bulgaria). *Folia Med. (Plovdiv)* 16(1):27-33, 1974.

1422 CHANGES IN URINARY γ-GLUTAMYLTRANSPEPTIDASE, LEUCINE AMINOPEPTIDASE, LACTATE DEHYDROGENASE, AND α-HYDROXYBUTYRATE DEHYDROGENASE IN HEPATOBILIARY DISEASE. *(Sp.)* Rodriguez Cuartero, A.; Nunez Carril, J. (San Cecilio Clin. Hosp., Granada, Spain). *Rev. Esp. Enferm. Apar. Dig.* 44(4):365-374, 1974.

1423 EFFECT OF A SINGLE DOSE OF FUROSEMIDE IN PATIENTS SUFFERING FROM CHRONIC HEPATIC DISORDERS. *(Sl.)* Dzurikova, V.; Brixova, E.; Niederland, T. R. (Res. Inst. Hum. Nutr., Conemius Univ., Bratislava, Czechoslovakia). *Bratisl. Lek. Listy* 61(3):336-343, 1974.

1424 HISTORIES OF PATIENTS WITH CHRONIC LIVER DISEASES IN A HEPATOLOGICAL SANATORIUM. *(Ger.)* Jorke, D.; Taeschner, H.; Wagner, M.; Reinhardt, M. (Jena Munic. Hosp., Germany). *Gesamte Inn. Med.* 29(14):580-587, 1974.

1425 CHRONIC HEPATITIS IN ADULTS AND CIRRHOSIS. *(It.)* Ventura, E. (Inst. Clin. Gen. Med. Med. Ther., Univ. Modena, Italy). *Med. Soc.* 23(2): 69-70, 1973.

1426 ONE CASE OF A SOLID MALIGNANT MESENTERIC TUMOR, CHRONIC HEPATITIS, AND CHYLOUS ASCITES DIAGNOSED BY LAPAROSCOPY. *(Sp.)* Herrerias Gutierrez, J. M.; Damiano Rivero, A.; Jimenez Peperez, J.; Sanchez Venegas, S.; Chamorro Lopez, J.; Garrido Peralta, M. (Fac. Med., Univ. Seville, Cadiz, Spain). *Rev. Esp. Enferm. Apar. Dig.* 44(3):257-266, 1974.

1427 PROBLEMS OF CHRONIC VIRAL HEPATITIS IN ADULTS AND THEIR SOCIAL IMPLICATIONS. *(It.)* Manenti, F. (Inst. Clin. Gen. Med. Med. Ther., Univ. Modena, Italy). *Med. Soc.* 23(2):65-69, 1973.

See also, 0888, 0915, 1196, 1318, 1380.

LIVER AND BILIARY TRACT

Alcoholic Liver Disease

1428 HIGH BLOOD ACETALDEHYDE LEVELS AFTER ETHANOL ADMINISTRATION: DIFFERENCE BETWEEN ALCOHOLIC AND NONALCOHOLIC SUBJECTS. *(E.)* Korsten, M. A.; Matsuzaki, S.; Feinman, L.; Lieber, C. S. (Bronx VA Hosp., N.Y.). *N. Engl. J. Med.* 292(8):386-389, 1975.

The blood acetaldehyde levels after experimental alcohol administration were investigated using 6 men with chronic alcoholism and 5 male control patients (aged 35-46 yr). In both alcoholic and control patients, blood acetaldehyde levels varied in a characteristic fashion in relation to ethanol. Acetaldehyde concentrations remained steady over a wide range of falling ethanol levels (from 54 to 33 mM), but abruptly dropped when the ethanol levels reached concentrations of 17.4 to 32.6 mM (mean 24 mM). Before this falloff, the acetaldehyde plateau of the alcoholic subjects fluctuated about a mean of 42.7 μM, significantly higher than that of the nonalcoholic, 26.5 μM. Peak ethanol levels and ethanol disappearance rates for the 2 groups were not significantly different. Losses of ethanol by respiratory and renal routes averaged 5% of the total amount infused (100 to 120 g). Percutaneous needle liver biopsies were performed on 2 alcoholic subjects. One biopsy revealed mild to moderate steatosis. The other demonstrated persistent lesions of alcoholic hepatitis, including alcoholic hyaline, fat and moderate inflammatory infiltrate. The results of this study show a dose-response relation between blood acetaldehyde and ethanol levels after ethanol administration. The ethanol concentration at which the fall of blood acetaldehyde occurred suggests desaturation of an ethanol oxidizing system other than alcohol dehydrogenase and indicates that at high ethanol blood levels, such a system contributes to ethanol oxidation. The higher acetaldehyde levels in alcoholism may result from both greater activity of this system and mitochondrial damage, and could contribute to the neurologic, hepatic and cardiac complications of alcoholism.

See also, 0905, 0909, 1042.

LIVER AND BILIARY TRACT

Cirrhosis

1429 HEPATOCYTE ULTRASTRUCTURAL CHANGES IN α_1-ANTITRYPSIN DEFICIENCY. *(E.)* Feldmann, G.; Bignon, J.; Chahinian, P.; Degott, C.; Benhamou, J. P. (INSERM, Beaujon Hosp., Clichy, France). *Gastroenterology* 67(6):1214-1224, 1974.

Liver cell ultrastructure was studied in 6 adult patients (3 male 3 female, 27-56 yr old) with α_1-antitrypsin deficiency; all except 1 were suffering from severe pulmonary emphysema. The liver was histologically normal in 3 patients, and portal fibrosis or cirrhosis was present in the other 3. Serum bilirubin, SGPT, BSP elimination, alkaline phosphatase, serum albumin, and γ-globulins were normal in 5 patients, except for SGPT in 1 patient which was 40 IU. In the sixth patient, serum albumin was 3.0 g/100 ml, alkaline phosphatase was 6.0 Bessey-Lowry-Brock U, and BSP retention at 45 min was 75%. Conventional electron microscopy showed specific changes consisting of deposits of an amorphous material in the dilated lumina of the endoplasmic reticulum; although these changes were demonstrated in all specimens, the number of liver cells showing these abnormalities varied markedly with each patient. The other liver cells were normal or were affected by nonspecific changes consisting mainly of an increased number of lysosomes. The liver cells with these nonspecific changes were seen only in the patients with liver fibrosis or cirrhosis. Electron microscopy, combined with the use of anti-α_1-antitrypsin antibodies labeled with horseradish peroxidase, demonstrated that a material antigenically similar

to α_1-antitrypsin was present in the deposits and on the ribosomes of the endoplasmic reticulum of those liver cells with the specific ultrastructural changes. It is suggested that this material is manufactured by these ribosomes, and that it accumulates in the endoplasmic reticulum lumina because of an abolished or reduced secretion. The fact that cirrhosis or fibrosis is absent in patients whose liver cells nevertheless contain deposits of this amorphous material supports the view that the ultrastructural hepatocytic lesions associated with, or leading to, fibrosis or cirrhosis are not directly related to accumulation of this material.

1430 ULTRASTRUCTURAL CHANGES OF JEJUNAL EPITHELIAL CELLS IN LIVER CIRRHOSIS. *(E.)* Portela-Gomes, G.; Martins, M. M.; Correia, J. P. (Univ. Hosp. St. Maria, Lisbon, Portugal). *Scand. J. Gastroenterol.* 9(7):657-663, 1974.

The stereomicroscopic and histological appearances of the jejunal mucosa and the ultrastructure of the villous absorbing-cells were studied in 10 patients with liver cirrhosis (9 chronic alcoholics and 1 non-alcoholic, 29-63 yr old) and in 5 normal subjects. Biopsies were performed with the Crosby-Kugler capsule under fluoroscopy. The controls had a normal structure and showed features similar to those previously published. In the 10 patients with cirrhosis normal finger and leaf-shaped villi were observed on the stereomicroscope. Histological examination of these biopsies showed a normal mucosa. In all cases, the ultrastructure of the epithelial cells showed an irregular pattern of abnormalities of varied severity. The abnormal cells showed irregularity and blistering of the microvilli, and the terminal web area was poorly developed, with an increased number of vacuoles. The mitochondria were swollen with clear spaces and myelin-like material. There were abundant vesicles containing lipid-like substance; the smooth endoplasmic reticulum was increased, and the rough endoplasmic reticulum persisted only in close association with mitochondria. There were enlarged vesicles of the Golgi complex. The nuclei were irregularly shaped, and in the cytoplasm there were autophagic vacuoles and myelin figures. The patient with cirrhosis and no alcoholism showed some alterations of the microvilli, autophagic vacuoles, and irregularly shaped nuclei. There was no clinical evidence of malabsorption in the patients, and the fecal fat excretion was normal in the 7 cases studied. No correlation of morphologic abnormalities with clinical findings was evident. The presence of numerous vesicles containing lipid-like substances suggests a delay in lipid transport through the cells. Ethanol-induced changes in the small intestine similar to those found in the 9 alcoholic patients with cirrhosis have been described in the literature. It is suggested that the observed abnormalities may be related to the toxic action of ethanol, because 7 of 9 alcoholic patients were in hospital for less than 30 days.

1431 α_1-ANTITRYPSIN DEFICIENCY, EMPHYSEMA, CIRRHOSIS, AND INTESTINAL MUCOSAL ATROPHY. *(E.)* Greenwald, A. J.; Johnson, D. S.; Oskvig, R. M.; Aschenbrener, C. A.; Randa, D. C. (Univ. Iowa Hosp., Iowa City). *JAMA* 231(3):273-276, 1974.

A case history is presented for a 64-yr-old man who had severe α_1-antitrypsin deficiency, pulmonary emphysema, hepatic cirrhosis, and a malabsorption syndrome indistinguishable from adult celiac disease. Laboratory investigations gave the following results: heamtocrit reading, 36%; leukocytes, 2,600/mm^3; serum iron concentration, 42 µg/100 ml; total iron-binding capacity, 270 µg/100 ml; serum carotene concentration 7 µg/100 ml; serum α_1-antitrypsin concentration, 50 mg/100 ml (normal, 180-244) as measured by radial immunodiffusion; total serum protein level, 7.5 g/100 ml; albumin level, 2.4 g/100 ml; alkaline phosphatase level, 101 mU/ml; SGOT concentration, 90 mU/ml; and total bilirubin, 0.9 mg/100 ml. The jejunal mucosal folds were thickened and the intestinal lumen was slightly dilated. A biopsy specimen of the jejunum demonstrated marked chronic inflammation in the lamina propria and villous atrophy, with disarray of the epithelial cells. In spite of a series of surgical procedures to correct gastrointestinal hemorrhaging, the patient continued to hemorrhage and died. Postmortem findings showed severe emphysema, portal cirrhosis, and a moderate amount of lymphocytic infiltrate in the liver. Numerous, round, periodic acid-Schiff (PAS)-positive, diastase-resistant intracytoplasmic inclusion bodies were seen within lobules varying in location from periportal to centrilobular with no clear predominance of either location. It is suggested that a celiac-like disease did not occur by chance in this case and that this condition may represent another variation among a group of diseases associated with low serum levels of α_1-antitrypsin. The physiologic function of α_1-antitrypsin in the human body is unknown, however, and the basis for an association between a celiac-like disease and α_1-antitrypsin deficiency has not been elucidated.

1432 PURIFICATION AND PARTIAL CHARACTERIZATION OF PAS-POSITIVE INCLUSION BODIES FROM THE LIVER IN ALPHA$_1$-ANTITRYPSIN DEFICIENCY. *(E.)* Eriksson, S.; Larsson, C. (Gen. Hosp., Malmo, Sweden). *N. Engl. J. Med.* 292(4):176-180, 1974.

The periodic acid-Schiff (PAS)-positive inclusion bodies were isolated from cirrhotic livers of patients who also had a primary hepatoma and homozygous α_1-antitrypsin deficiency and their main component, hepatic antitrypsin, partially characterized. Portions of livers weighing 30 g were cut into thin slices which were incubated for 1 hr at 37 C with 50 ml Hanks buffer containing 25 mg collagenase and 0.02% sodium azide. The filtrate was centrifuged and the resulting pellet taken up in 12 ml 0.25 M sucrose containing 0.02% sodium azide. The suspension was sonicated for 30 sec before undergoing gradient centrifugation. The final pellet consisted of practically pure PAS-positive globules of uniform size with an average diameter of 2-3 µm, approximately the same molecular size as serum α_1-antitrypsin. Immunologic similarity between the isolated protein and serum α_1-antitrypsin was established by the isolate's ability to react with

fluorescein-isothiocyanate-conjugated antibodies against serum α_1-antitrypsin and to raise antibodies against it in rabbits, and with the double immunodiffusion technique. On agarose gel electrophoresis hepatic antitrypsin migrated as an α_2-globulin before and after neuraminidase treatment. Chemical analysis showed a complete absence of sialic acid. Hepatic antitrypsin had a pronounced tendency to form insoluble macroaggregates. The author concludes that the available data support the concept that the accumulation of antitrypsin in the endoplasmic reticulum is associated with an insufficient sialylation of the protein.

1433 A STUDY OF CARBOHYDRATE METABOLISM IN POSTNECROTIC CIRRHOSIS OF LIVER. *(E.)* Yeung, R. T. T.; Wang, C. C. L. (Queen Mary Hosp., Hong Kong). *Gut* 15(11):907-912, 1974.

The relationship between carbohydrate metabolism and insulin secretion was studied in 21 patients (all male, 23-58 yr) with postnecrotic cirrhosis of undetermined origin and the disorders of carbohydrate metabolism were correlated with liver function. I.v. glucose tolerance, insulin tolerance, tolbutamide, and glucagon tests were performed. Based arbitrarily on the BSP retention test the patients were divided into group A, 9 patients with less impaired liver function, and group B, 12 patients with greater impairment of liver function. I.v. glucose and insulin tolerances were reduced in both groups. The hypoglycemic effect of tolbutamide was similar in the controls (10 healthy male subjects) and in both groups of cirrhotic patients, but this was achieved at higher plasma insulin levels in group B indicating resistance of the liver to the effect of endogenous insulin. The blood glucose response to glucagon was markedly impaired in group B patients which is consistent with this hypothesis. The insulin response to glucagon was reduced in the cirrhotic patients. Fasting human growth hormone and free fatty acid levels were elevated in both groups, but statistical analysis showed no significant correlation between plasma growth hormone or free fatty acid levels and the production of insulin resistance. It is concluded that impaired glycogenesis accounts for insulin resistance in a proportion of cases only and that other factors have yet to be identified.

1434 ASSESSMENT OF AMINOPYRINE METABOLISM IN MAN BY BREATH ANALYSIS AFTER ORAL ADMINISTRATION OF ^{14}C-AMINOPYRINE: EFFECTS OF PHENOBARBITAL, DISULFIRAM AND PORTAL CIRRHOSIS. *(E.)* Hepner, G. W.; Vesell, E. S. (Pennsylvania State Univ. Coll. Med., Hershey). *N. Engl. J. Med.* 291(26):1384-1388, 1974.

Fifteen healthy ambulant control subjects (11 male, 4 female, 21-80 yr), 10 healthy nonambulant controls (5 male, 5 female, 23-66 yr), and 10 patients with portal cirrhosis (7 male, 3 female) were given 2 μCi ^{14}C-aminopyrine p.o. together with 9 mg/kg unlabeled aminopyrine. Blood samples of 10 ml were taken 3, 6, 9, and 12 hr after aminopyrine administration. Breath samples were collected hourly, 1-8 hr and 12 hr after drug administration. Some of the volunteers were restudied 2 weeks after the 1st study, after 7 days pretreatment with phenobarbital (2 mg/kg/day), or after 4 days pretreatment with disulfiram (250 mg/day). Aminopyrine in serum samples was analyzed spectrophotometrically; breath samples were counted in a liquid scintillation counter. Aminopyrine metabolic clearance rate and 12-hr breath $^{14}CO_2$ output in the healthy volunteers studied before and after drug pretreatment correlated highly ($r = 0.91$). The metabolic clearance rate was 46 ml/min in the patients with portal cirrhosis, and 122 ml/min in the healthy controls. $^{14}CO_2$ breath output at 12 hr was 13.8% in the patients with portal cirrhosis, and 32.6% in the nonambulant controls. It is suggested that *in vivo* breath analysis after administration of ^{14}C-aminopyrine provides useful information about hepatic drug metabolism in health and disease.

1435 IRON STORAGE DISORDERS OF THE LIVER. *(E.)* Grace, N. D.; Powell, L. W. (Tufts Univ. Sch. Med., Boston, Mass.). *Gastroenterology* 67(6): 1257-1283, 1974.

An extensive review of the literature on hepatic disorders associated with increased iron deposition is presented. Hemochromatosis is defined as a pathological entity with the following characteristics: cirrhosis or fibrosis of the liver with increased iron stores, primarily in the parenchymal cells of the liver but also in other organs, including pancreas, heart, pituitary, and adrenals. It includes all of the following: idiopathic hemochromatosis, cirrhosis of the liver with secondary iron overload, liver disease associated with certain anemias, with dietary iron overload, or with transfusional, and congenital transferrin deficiency. The following aspects of iron metabolism are discussed in relation to the development of hepatic iron overload: absorption, body content and distribution, transport, storage, and mobilization. Acute and chronic iron toxicity are considered as are the pathology and diagnosis of hemochromatosis. Each of the disease entities included under the definition of hemochromatosis is treated in some detail, including their prognosis and management. A number of conclusions were reached as a result of this review: 1) hemochromatosis is not a single disease but rather should be viewed as several disorders leading to a similar pathological entity. There is an apparent difference in the geographical distribution of these disorders; 2) clinical evidence suggests that iron is at least a contributing factor in the development of hepatic injury; 3) if phlebotomy treatment of idiopathic hemochromatosis is employed, the patient should be maintained in an iron-depleted state by repeated phlebotomy; 4) phlebotomy does not appear to have a role in the treatment of the alcoholic cirrhotic with excess iron stores; 5) patients with idiopathic hemochromatosis can be differentiated from the alcoholic cirrhotic with increased hepatic iron stores by a strong family history of iron storage disease, lack of a history of increased alcohol intake, and significant cardiac or endocrine involvement, especially of the pituitary. Three hundred references are cited.

1436 FIBRINOGEN IN LIVER DISEASE. *(E.)* Wardle, E. N. (R. Victoria Infirm., Newcastle upon Tyne, England). *Arch. Surg.* 109(6):741-746, 1974.

Plasma fibrinogen catabolism was studied by a ^{125}I-fibrinogen assay in 42 patients with liver disease (liver cirrhosis, trauma, acetaminophen toxicity, biliary obstruction, metastatic liver disease, cholangitis or cholestatic hepatitis) and in 24 controls. All patients received p.o. sodium iodide to block the thyroid, and plasma and urinary fibrinogen levels were determined at various times following injection of ^{125}I-fibrinogen. The fibrinogen half-life in controls was 95.3 hr with a fractional catabolic rate (percent of the intravascular pool catabolized per day) of 21.8%. The highest fibrinogen catabolism rates were found in patients with cholangitis (half life = 42.5-65 hr) and liver contusion (36-52 hr). Increased fibrinogen catabolism also occurred with portal cirrhosis (half life = 53.6 hr; fractional catabolic rate = 39.6%) and in obstruction (51.0-67.2 hr; 34.2-43.5 hr). Portal cirrhosis patients had increased fibrinolysis while obstructed patients showed decreased fibrinolysis. The increased fibrinogen catabolism could be accounted for by liver cell necrosis and, more commonly, by endotoxemia or overt infection. Endotoxin assays showed a correlation between increased endotoxin levels and the presence of intravascular coagulation suggesting that endotoxemia plays an important role in the intravascular coagulation associated with liver disease.

1437 ISOPROTERENOL IN OFFSETTING ADVERSE EFFECTS OF VASOPRESSIN IN CIRRHOTIC PATIENTS. *(E.)* Sirinek, K. R.; Thomford, N. R. (Ohio State Univ. Coll. Med., Columbus). *Am. J. Surg.* 129(2):130-136, 1975.

The combined administration of isoproterenol and vasopressin was investigated in 10 patients with Laennec's cirrhosis and portal hypertension. The portal vein, superior vena cava and one radial artery were catheterized for continuous monitoring of pressures and cardiac output. Forty-eight to 72 hr after catheterization, each patient was put through a daily study for 3 days consisting of a 60 min stabilization period, a 60 min test period, and a 60 min observation period. During the test period, a 4 U/ml 5% dextrose solution of lysine-vasopressin was infused into a vein in the arm at 100 ml/hr for 1 hr. Those who had a significant drop in cardiac output were studied for a second 3 day period using in addition to the previous dosage of vasopressin, a 0.002 mg/ml 5% dextrose solution of isoproterenol. During the first 3 day period, the portal pressures decreased on the average from 354 to 248 mm saline after 60 min. Five of the 10 patients had also had a significant reduction of cardiac output from 6.6 to 4.7 liter/min within 5 min of infusion. When these patients were given both drugs, cardiac output remained close to control value. Reduction in portal pressure in this group was from 371 to 271 mm saline after 60 min. It is concluded that isoproterenol may reduce the hemodynamic hazards associated with administration of vasopressin.

1438 SPLENOAZYGOS SHUNT FOR STENOSIS OF THE SUPRAHEPATIC INFERIOR VENA CAVA WITH CIRRHOSIS: SECONDARY BUDD-CHIARI SYNDROME. *(E.)* Saubier, E. C.; Brault, A.; Partensky, C. (no affil.). *Int. Surg.* 59(10):526-532, 1974.

A 30 yr old North African male was hospitalized for ascites and vomiting. There was a previous hospitalization for ascites from extrahepatic disease. Examination disclosed stenosis of the inferior vena cava with collateral circulation but no compensation of portal hypertension. There were esophageal varices from the cardia to the aortic arch, enlargement of the spleen, cirrhosis and secondary Budd-Chiari syndrome, indicated by obstruction of the supra-hepatic veins. Surgery was performed to direct the portal flow into the superior vena cava by construction of a splenoazygous anastomosis. A 5 month follow-up showed the collateral circulation had disappeared and the esophageal varices had decreased.

1439 SIDE-TO-SIDE PORTACAVAL SHUNT IN THE TREATMENT OF BUDD-CHIARI SYNDROME. *(E.)* Prandi, D.; Reff, B.; Benhamou, J. P. (INSERM, Beaujon Hosp., Clichy, France). *Gastroenterology* 68(1):137-141, 1975.

A 22 yr old woman was hospitalized for ascites, and a diagnosis of Budd-Chiari syndrome of unknown etiology was made. Because attempts at control by low sodium diet, furosemide, and spironolactone were unsuccessful, surgery was indicated. Ascitic fluid was removed during laparotomy. The right lobe of the liver was atrophic while the left was enlarged and purplish, and the Spigel lobe was slightly enlarged. A side-to-side portacaval shunt was constructed. The patient's condition improved rapidly after surgery and liver size slightly decreased. Her condition was still excellent 19 months later. It is suggested that side-to-side portacaval shunt be considered in patients with recurrent or intractable ascites.

1440 PORTACAVAL ANASTOMOSIS AND PEPTIC ULCER: A NONASSOCIATION. *(E.)* Phillips, M. M.; Ramsby, G. M.; Conn, H. O. (VA Hosp., West Haven, Conn.). *Gastroenterology* 68(1):121-131, 1975.

Cirrhotic patients with portacaval anastomoses were compared with unshunted cirrhotic patients to determine whether the operation results in greater frequency of peptic ulcer. One hundred eight subjects were divided into 2 randomized groups of which 48 had prophylactic portacaval anastomoses and 60 were unoperated controls. To establish the clinical relevance of the randomized groups, 2 non-randomized groups were included, of which 77 were excluded from the randomized study and 44 had been given therapeutic portacaval anastomoses. Peptic ulcer diagnosis was based on demonstration of the crater by x-ray, endoscopy or surgery. There were 2 statistically significant findings. (1) Eleven of the 60 controls, and 11 of the 48 with prophylactic anastomoses had had peptic ulcers at some time. (2) In the combined unoperated groups, there was an

average of 19 months between inclusion in the study and diagnosis of ulcer, while in the combined operated groups it was 48 months. In a 2 yr inclusion period 76% of the post-inclusion ulcers occurred in the unoperated groups while in the combined operated groups only 38% occurred, suggesting that the operation delays clinical appearance of peptic ulcer. It is concluded that the prevalence of peptic ulcer in cirrhotic patients increases progressively with time, regardless of surgery.

1441 THORACIC DUCT AND HEPATIC LYMPH IN IDIOPATHIC PORTAL HYPERTENSION. *(E.)* Samanta, A. K. S.; Saini, V. K.; Chhuttani, P. N.; Patra, B. S.; Vashista, S.; Datta, D. V. (Postgrad. Inst. Med. Educ. Res., Chandigarh, India). *Gut* 15(11): 903-906, 1974.

Lymph dynamics were studied in 2 phases in idiopathic portal hypertension: 1) the thoracic duct lymph was studied in 11 patients by cannulating the duct to determine if there is any alteration in lymph transport; and 2) the hepatic lymphatics were studied by percutaneous hepatography in 16 patients. In addition, the hepatic lymph was studied in 13 other cases of cirrhosis of the liver and in 5 control subjects. Patients with idiopathic portal hypertension and cirrhosis had bleeding from varices in the recent past. Parameters studied were thoracic duct size and pressure, lymph flow rate, hematocrit, and protein. The thoracic duct was appreciably greater in diameter, distended, and turgid with lymph in patients with idiopathic portal hypertension. The lymph flow rate (2.70 ml/min) was consistently higher than the normal of 1 ml/min. Patients with cirrhosis of the liver had a mean lymph flow rate of 3.44 ml/min. Analysis of the lymph flow rate revealed no statistically significant correlation with the serum albumin and the 45-min plasma BSP retention. There was a positive, but not statistically significant, correlation with such hemodynamic parameters as free hepatic venous pressure, wedge hepatic venous pressure, estimated hepatic blood flow, and hepatic vascular resistance. The mean lymph:serum protein ratio was markedly lower in idiopathic portal hypertension (0.29) than in cirrhosis (0.41). Lymph was grossly hemorrhagic in all patients with idiopathic portal hypertension and the lymph hematocrit was 1.45%. Hepatic lymphatics were demonstrated in 3 of 16 patients studied by percutaneous hepatography. The lymphatics appear as tortuous and beaded interlacing channels traversing the liver towards its hilum and finally draining to the paraaortic region. No single hemodynamic parameter could be solely incriminated in the pathogenesis of increased liver lymph formation. The finding of hemorrhagic lymph suggests the hepatic sinusoidal bed as one of the sources of increased thoracic duct lymph. The fact that the lymph:serum protein ratio was distinctly lower in idiopathic portal hypertension than in cirrhosis may indicate that the contribution of intestinal lymph to the thoracic duct in idiopathic portal hypertension is a more important factor than in cirrhosis.

1442 COEXISTENT INFERIOR VENA CAVAL AND PORTAL HYPERTENSION. SPLENIC TO LEFT HEPATIC VEIN ANASTOMOSIS IN A CIRRHOTIC PATIENT. *(E.)* Maillard, J. N.; Hay, J. M.; Elman, A.; Sicard, J. L. (Louis Mourier-UER Xavier-Bichat Hosp., Colombes, France). *Arch. Surg.* 109(6):819-821, 1974.

A portal-systemic shunt designed to decompress the splanchnic venous circulation when the inferior vena cava is not available was used in a patient with a preoperative diagnosis of combined portal and caval hypertension. The patient, a 44 yr old man with cirrhosis presumably due to alcoholism, had undergone an end-to-side portacaval shunt 1 yr earlier. In the present surgery, the patient underwent splenectomy, left lateral hepatic lobectomy, and end-to-end anastomosis of the splenic vein to the left hepatic vein which was dissected free to its juncture with the inferior vena cava at the diaphragm. An end-to-end anastomosis 20 mm in diameter was then possible with a flow through this shunt of 900 ml/min. The portal pressure, however, was reduced only from 40 to 30 mm Hg. The persistent high portal pressure was attributed to over transfusion. The postoperative course was complicated but by the 50th postoperative day, the patient was well enough to be restudied. An arteriogram showed that blood flowed predominantly from the superior mesenteric vein retrograde through the splenic vein and the splenohepatic shunt; enough contrast material went through the portacaval shunt to visualize the narrow retrohepatic segment of the inferior vena cava and the small hepatic portion of the portal vein. Subsequently, the patient continued to recover slowly, but 4 months postoperatively died from liver failure secondary to pulmonary sepsis. The authors claim that the operation has several advantages over those previously described: 1) it does not require a combined thoracic and abdominal approach; 2) it involves only a single vascular anastomosis and does not require a prosthetic conduit; and 3) the shunt fits the pathologic anatomy nicely, and is capable of handling a large flow volume. It is indicated when portal hypertension must be corrected in the face of extrinsic retrohepatic caval compression or thrombosis.

1443 NODULAR, NON-CIRRHOTIC LIVER ASSOCIATED WITH PORTAL HYPERTENSION IN A PATIENT WITH RHEUMATOID ARTHRITIS. *(E.)* Harris, M.; Rash, R. M.; Dymock, I. W. (Dep. Pathol., Univ. of Manchester, England). *J. Clin. Pathol.* 27(12):963-966, 1974.

A case is presented of liver nodularity associated with rheumatoid arthritis but not with Felty's syndrome. A 79 yr old woman presented with massive ascites, ankle and sacral edema, and gangrene of 2 fingertips. There was inactive rheumatoid arthritis. The ascites was tapped, but attempts to keep it under control with diuretics failed, and the patient died of liver failure shortly thereafter. Diagnosis was hepatic cirrhosis with portal hypertension and bleeding from esophageal varices. Necropsy revealed an unusually small liver (592 g) with an ill-defined nodularity on the surface and evidence of portal hypertension. Light microscopy showed the nodularity to be due to changes in the texture and arrangement of the liver cell plates. This resembled 5 previous cases in which nodularity without fibrosis was

associated with portal hypertension; however, this was the first case in which Felty's syndrome was not present. It is suggested that the condition may be associated with the rheumatoid process *per se* rather than Felty's syndrome.

1444 HEPATIC INJURY FROM CHRONIC HYPERVITAMINOSIS A RESULTING IN PORTAL HYPERTENSION AND ASCITES. *(E.)* Russell, R. M.; Boyer, J. L.; Bagheri, S. A.; Hruban, Z. (Dep. Med., Univ. Chicago, Ill.). *N. Engl. J. Med.* 291(9):435-440, 1974.

Case histories are presented of 2 patients with chronic vitamin A intoxication caused by massive doses of the vitamin taken for 5-8 yr because of health fetishes. The patients were initially seen because of ascites and clinical signs of cirrhosis. Histologic examination of liver biopsy material revealed perisinusoidal fibrosis, central-vein sclerosis and focal congestion associated with perisinusoidal lipid storage cells. Vitamin A fluorescence was found on frozen sections, and direct assay of tissue revealed substantial increases in vitamin A content (1700 to 2200 µg/g wet liver). Electron microscopy demonstrated an increase in basement-membrane-like material and collagen within the perisinusoidal space in association with lipid-filled Ito cells. The nature of this unusual lesion suggested that it might obstruct hepatic blood flow, result in considerable parenchymal-cell atrophy and lead to portal hypertension. It is further suggested that chronic ingestion of large amounts of vitamin A stimulates fibrogenesis and results in a disorder of hepatic function resembling cirrhosis. Ascites has remained a continuing problem with one of the patients, even though vitamin A was stopped. It is possible that the stimulus to fibrosis may continue since mobilization of the large hepatic stores of vitamin A may take years.

1445 ULTRASTRUCTURAL ASPECTS OF PRIMARY BILIARY CIRRHOSIS AND OTHER TYPES OF CHOLESTATIC LIVER DISEASE. *(E.)* Chedid, A.; Spellberg, M. A.; DeBeer, R. A. (Univ. Cincinnati Sch. Med., Ohio). *Gastroenterology* 67(5):858-869, 1974.

Liver biopsies performed in 7 patients with primary biliary cirrhosis, 5 with extrahepatic obstructive jaundice, 3 with cholestatic viral hepatitis, 2 with chronic aggressive hepatitis, and 2 with other types of cirrhosis, were studied by light and electron microscopy. Serial liver biopsies were available in the 7 patients with primary biliary cirrhosis and these were studied at different stages of the disease. Distinctive features were observed in patients with primary biliary cirrhosis in the small and medium-sized bile ducts. The bile duct epithelium showed intracytoplasmic filamentous structures grouped into bundles which became progressively more abundant and prominent. The mitochondria were swollen and showed fewer circular cristae; the cytoplasm in addition was occupied by numerous cytophagosomes which decreased with the progression of the disease. The rough endoplasmic reticulum was vesicular and ribosomes were few. The luminal microvilli were at first blunted and scanty and finally disappeared. In the final stage, the basement membranes of the bile ducts were disrupted and invaded by polymorphonuclear leukocytes. Changes were observed in hepatocytes as well. At the early stage they contained numerous cytophagosomes, and the mitochondria showed homogenization of their matrix with curling and disappearance of cristae, granules, and prominent crystalline inclusions. These hepatocyte findings vary with the progression of the disease and do not appear to be characteristic. Some of the changes observed in the bile ducts are also seen to a minor degree in other pathological entities, but their prominence and progression are distinctive in primary biliary cirrhosis and suggest that they may be features of possible diagnostic significance when biochemical, clinical, or histological criteria are inconclusive or equivocal.

1446 COPPER AND PRIMARY BILIARY CIRRHOSIS. *(E.)* Fleming, C. R.; Dickson, E. R.; Baggenstoss, A. H.; McCall, J. R. (Mayo Clin., Rochester, Minn.). *Gastroenterology* 67(6):1182-1187, 1974.

In an autopsy study, hepatic copper concentrations in primary biliary cirrhosis (8 cases) were compared with those in normal organs (10 subjects), in postnecrotic cirrhosis (15 cases), in alcoholic cirrhosis (13 cases), and in extrahepatic obstruction (10 cases). Tissue copper stores in the spleen, the renal cortex, and the cerebral cortex also were assessed in primary biliary cirrhosis and normals. All copper measurements were done by atomic absorption spectrophotometry. Histological procedures in which hematoxylin and eosin and van Gieson stains were used provided evidence for the presence or absence of regenerative nodules and for the degree of fibrosis. The mean hepatic copper level (441 µg Cu/g dry weight) in primary biliary cirrhosis was significantly greater than that in normal groups (27 µg Cu/g dry weight) and in both groups with cirrhosis (postnecrotic: 57 µg Cu/g dry weight; alcoholic: 13 µg Cu/g dry weight). When multiple random-coded needle biopsy specimens for hepatic copper were obtained in each case of primary biliary cirrhosis, there was only modest variation in copper distribution among specimens, suggesting that a single biopsy adequately reflects elevated Cu levels. In extrahepatic obstruction, hepatic copper levels appeared to increase directly with the duration of obstruction. Compared to normal subjects, patients with primary biliary cirrhosis had greater copper levels in the spleen and renal cortex with normal or only minimally elevated cerebral cortex copper levels. It is suggested that these elevated levels of copper in other major organs probably represent saturation of hepatic stores, overflow into the plasma, and transportation of albumin-bound copper to other tissue depots, and that primary biliary cirrhosis is associated with a more generalized disorder of copper metabolism than was previously thought.

1447 FUNCTIONAL RENAL FAILURE AND HEMODYNAMIC ABNORMALITIES OF CIRRHOSIS. A REVIEW. *(Gr.)* Manesis, E. K. (no affil.). *Iatriki* 23(5):381-393, 1973.

1448 EMERGENCY PORTACAVAL SHUNTS. *(Fr.)* Bergeron, C.; Roberge, R. (Chicoutimi Hosp., Canada). *Union Med. Can.* 103(9):1564-1572, 1974.

1449 CONTROLLED ARTERIALIZATION OF THE LIVER. II. CLINICAL RESULTS. *(Ger.)* Fritsch, A.; Funovics, J.; Gangl, A.; Horak, W.; Grabner, G.; Koha, P. (1st Surg. Clin., Univ. Vienna, Austria). *Langenbecks Arch. Chir.* 336(1):67-89, 1974.

1450 SURGERY FOR PORTAL HYPERTENSION AND LIVER REGENERATION. *(Por.)* Ribeiro, E. B. (Sao Lucas Sanit., Sao Paulo, Brazil). *Bol. Sanatorio Sao Lucas* 35(5):89-91, 1974.

1451 HAEMATEMESIS IN THE AFRICAN: A COMPARISON OF THE TWO MAIN CAUSES AFTER A TEN YEAR INTERVAL. *(E.)* Gelfand, M. (Harare Cent. Hosp., Rhodesia). *Cent. Afr. J. Med.* 20(5):100-101, 1974.

1452 POSTOPERATIVE FIBRINOLYTIC BLEEDING IN CIRRHOSIS. *(Ger.)* Aschauer, R.; Kunz, F.; Constantini, R.; Holzknecht, F. (Med. Clin., Univ. Innsbruck, Austria). *Wien. Klin. Wochenschr.* 86(15):436-438, 1974.

1453 SEX, ASCITES AND ALCOHOLISM IN SURVIVAL OF PATIENTS WITH CIRRHOSIS: EFFECT OF PREDNISONE. *(E.)* Juhl, E.; Winkel, P.; Tygstrup, N. (Univ. Hosp. Copenhagen, Denmark). *Digestion* 10(4/5):310, 1974.

1454 AROUSAL RESPONSE IN HEPATIC ENCEPHALOPATHY WITH L-DOPA. *(E.)* Weiss, A.; Pitman, E. R.; Javdan, P. (Mt. Sinai Hosp. Serv., Elmhurst, N.Y.). *Am. J. Gastroenterol.* 62(6):497-503, 1974.

1455 CHANGES IN LABORATORY FINDINGS IN CHRONIC DISEASES OF THE LIVER DURING INTERMITTENT TREATMENT WITH PURINOR. *(Cz.)* Ondrejicka, M.; Mikulecky, M.; Hantak, I.; Kratochvilova, H. (1st Med. Fac., Comenius Univ., Bratislava, Czechoslovakia). *Bratisl. Lek. Listy* 61(4):413-427, 1974.

1456 IT IS POSSIBLE TO CLINICALLY CURE CIRRHOSIS IN A LARGE PERCENTAGE OF CASES BY TREATMENT WITH LARGE DOSES OF TESTOSTERONE AND VITAMIN B_1. *(It.)* Girolami, M. (Inst. Clin. Trop. Infect. Dis., Univ. Rome, Italy). *Clin. Eur.* 12(4):301-315, 1974.

1457 HYPOCALCEMIA IN CIRRHOSIS. *(Fr.)* Darnis, F.; Cambier, J.; Masson, M.; Lhuillier, M.; Mosse, A. (Beaujon Hosp., Clichy, France). *Ann. Med. Interne (Paris)* 125(5):475-480, 1974.

1458 CLINICAL FORMS AND PATHOGENY OF BANTI'S DISEASE. *(Fr.)* Varay, A. (Rothschild Hosp., Paris, France). *Ann. Gastroenterol. Hepatol. (Paris)* 10(3):227-240, 1974.

1459 INSULIN SECRETION IN HAEMOCHROMATOSIS: ISOLATED ALTERATION OF THE RESPONSE TO GLUCOSE STIMULUS PER OS IN THE EARLY STAGES. *(E.)* Hespel, J. P.; Simon, M.; Lecornu, M.; Bourel, M. (INSERM, Pontchaillou Hosp., Rennes, France). *Digestion* 10(4/5):355, 1974.

See also, 1127, 1146, 1197, 1198, 1199, 1321, 1338, 1341, 1402, 1418, 1423, 1424, 1425.

LIVER AND BILIARY TRACT

Gallbladder and Biliary Tract Diseases

1460 EFFECT OF DIFFERENT DOSES OF CHENODEOXYCHOLIC ACID ON BILE-LIPID COMPOSITION AND ON FREQUENCY OF SIDE-EFFECTS IN PATIENTS WITH GALLSTONES. *(E.)* Mok, H. Y. I.; Bell, G. D.; Dowling, R. H. (R. Postgrad. Med. Sch., London, England). *Lancet* (7875):253-257, 1974.

The effect of various doses of chenodeoxycholic acid (CDA) (250, 500, 750, and 1000 mg/day) on bile-lipid composition and the frequency of side-effects were studied in 38 patients (30 women and 8 men, ranging in age from 22-75 yr) with gallstones. At a dose of 250 mg/day, CDA therapy did not reduce cholesterol saturation of bile in 3 of 7 patients, but treatment with 500 mg/day or more led to a progressive desaturation of bile with cholesterol. While the response of bile-lipid composition to CDA in individual patients was variable, the overall pattern of results suggested that the higher the dose, the greater the reduction in the saturation of bile with cholesterol. Diarrhea was present in 40% of patients taking between 750-1000 mg CDA/day; at these doses SGOT was found to be slightly raised on at least one occasion during monthly checks in 7 of 27 patients. Other studies of liver function and structure, however, were normal, irrespective of dose. On withdrawal of CDA therapy, bile-lipid composition reverted to its supersaturated state, suggesting that once gallstones have been dissolved, long-term maintenance or intermittent CDA therapy may be necessary.

1461 ACCELERATING GALLSTONE DISSOLUTION. *(E.)* Tao, J. C.; Cussler, E. L.; Evans, D. F. (Dep. Chem. Eng., Carnegie-Mellon Univ., Pittsburgh, Pa.). *Proc. Natl. Acad. Sci. USA* 71(10):3917-3921, 1974.

Three experimental methods were used to study the mechanisms responsible for cholesterol gallstone dissolution in bile. The model bile solutions used in these experiments contained various amounts of bile salts, lecithin, cholesterol, and sodium chloride. Discs and spheres of radioactive cholesterol were used as were real gallstones (white or pale yellow spheres) containing more than 90% cholesterol. Dissolution was determined by liquid scintillation counting. The first method, used for rapidly flowing bile, involved a spinning disc of cholesterol immersed in a large volume of bile solution. The second experimental method used a cholesterol sphere or a real gallstone suspended in bile. For studies at high flow, the sphere was suspended in a tube through which bile was steadily pumped at a known rate. For studies at low flow, where free convection is important, the sphere was suspended in an unstirred beaker of bile. The third experimental apparatus, used only for studies in stagnant bile, consisted of a cholesterol slab mounted at the bottom of a cylindrical volume of bile solution. This volume was bounded by identical holes in 4 flat plates, which could be slid apart without causing flow in the vertical direction. The dissolution rates of cholesterol were controlled by diffusion in slowly flowing bile and by interfacial kinetics in rapidly flowing bile. At low flow, dissolution varied with the square root of bile flow and could be predicted, *a priori*, from existing correlations of mass transfer. At high bile flow, dissolution was independent of bile flow and probably dominated by the rate of micelle adsorption. These results indicate that cholesterol gallstone dissolution can be accelerated little in slow bile, but more significantly in rapidly flowing bile.

1462 CHOLEDOCHAL CYST: A REVIEW OF 18 CASES. *(E.)* Yue, P. C. K. (Queen Mary Hosp., Hong Kong). *Br. J. Surg.* 61(11):896-900, 1974.

Eighteen cases of choledochal cyst affecting Chinese patients (15 female and 3 male, 53 days to 37-yr old) seen during an 18-yr period are reported. The clinical picture was 1 of 4 types: jaundice as the only feature in infancy; abdominal pain, jaundice and an abdominal mass or an abdominal mass without jaundice, in children and adults; bleeding from esophageal varices due to biliary cirrhosis in terminal cases. A correct preoperative diagnosis was made in 13 cases. Barium meal examination was carried out in 10 and all showed diagnostic features. I.v. cholangiography was performed in 7 cases. The biliary tree was not visualized in 2 cases; in 5 the choledochal cyst and the rest of the biliary tract were outlined. Seventeen patients underwent surgery: 3 had choledochocystojejunostomy, 4 had choledochocystoduodenostomy and 10 had cyst excisions. Three of the 18 patients died, 2 of them after surgery. Five patients had leakage after surgery, 2 being those who died, the other 3 being successfully treated with parenteral nutrition combined with surgery. Of the 12 patients available for follow-up (6 months to 5 yr postoperatively), 2 have had recurrent cholangitis but the other 10 are well. The incidence of choledochal cysts represented by this study is 1 per 5000 hospital admissions, suggesting that the Chinese are more often affected than occidentals among whom the incidence is reported as 1 in 260,000 admissions and 1 in 13,000 admissions in two previously published reports.

1463 THE RESULTS OF CHOLECYSTOSTOMY FOR THE TREATMENT OF ACUTE CHOLECYSTITIS. *(E.)* Gagic, N.; Frey, C. F. (Wayne Cty. Gen. Hosp., Ann Arbor, Mich.). *Surg. Gynecol. Obstet.* 140(2):255-257, 1975.

Twenty-two cholecystostomies were reviewed to determine whether misuse of the operation accounts for its high mortality rate. Twenty-one patients presented with pain and tenderness in the right upper abdominal quadrant, 7 had a palpable mass, and 6 had jaundice. Cholecystostomy rather than cholecystectomy was performed due to age of patient, to technical difficulties due to inflammation, to gram-negative sepsis, or to presence of renal, cardio-vascular, hepatic or pulmonary disease. All were found to have acute cholecystitis on surgery. Six of the 22 patients died, all from a combination of ascending cholangitis and gram-negative sepsis. It is suggested that survival in ascending cholangitis depends on immediate and adequate bile duct drainage, which is not provided by the operation as performed. It is concluded that the deaths resulted from misuse of the operation, and could have been avoided by use of T-tube drainage of the common bile duct.

1464 BILIARY PERITONITIS WITHOUT PERFORATION OF THE GALLBLADDER IN ACUTE CHOLECYSTITIS. *(E.)* Kent, S. J. S.; Menzies-Gow, N. (St. Thomas's Hosp., London, England). *Br. J. Surg.* 61(12):960-962, 1974.

Six cases of biliary peritonitis complicating acute cholecystitis were studied. Five of these had no demonstrable gallbladder perforation, however the gallbladder was either distended, ischemic or both. The average age of the patients was 73, suggesting that the ischemia may have been augmented by arteriosclerosis or low cardiac output. It is concluded that ischemia caused by distension may cause the gallbladder mucosa and overlying peritoneum to become permeable to bile.

1465 BILE PERITONITIS—A REPORT OF FIFTEEN PATIENTS. *(E.)* Ellis, H.; Adair, H. M. (Westminster Hosp., London, England). *Postgrad. Med. J.* 50(589):713-717, 1974.

Fifteen patients with bile peritonitis are reported with regard to etiology, clinical picture, prognosis, and treatment, and the current literature is reviewed. Of the patients, one suffered traumatic rupture of the gallbladder, 6 had acute cholecystitis, 7 had postoperative leaks, and 1 had a leak after T-tube removal. The clinical picture correlated somewhat with the broad etiological categories. Those cases stemming from perforation in acute cholecystitis presented with widespread peritonitis and acute pain. The non-specific signs and symptoms and the

condition's rarity made diagnosis difficult. Those cases resulting postoperatively occasionally presented more insidiously with mild pain and tenderness. Mortality was typically high (40%). It is suggested that the high mortality is related to the age of the patient, length of history, and promptness of treatment. It is also suggested that pre-operative bile peritonitis be treated surgically. In the postoperative leaks laparotomy is indicated.

1466 PRURITIC EFFECT OF BILE SALTS. *(E.)* Kirby, J.; Heaton, K. W.; Burton, J. L. (Bristol R. Infirm., England). *Br. Med. J.* 4(5946):693-695, 1974.

Nine purified bile salts were tested for their ability to produce pruritus in 24 volunteers. The sodium salts of cholic, deoxycholic and chenodeoxycholic acids and their taurine and glycine conjugates were each dissolved in phosphate buffered saline (1 mM/liter); the deoxycholate and chenodeoxycholate acids were first dissolved in 5% sodium bicarbonate. In 6 subjects, conventional patch and prick tests of each solution were made. In 20, double blind, randomized blister base tests were made by injection of each solution for 2 min into cantharidin-induced blisters. Normal saline solution and papain diluted 1/10 were used as controls. The patch and prick tests were entirely negative. The blister base test, in contrast, caused pruritus with all solutions in at least 2 subjects, within 45 sec of injection. The dihydroxy salts were effective in 56 of 120 tests and the trihydroxy salts 11 out of 60, a significant difference that was even greater with unconjugated rather than conjugated salts. The saline solution caused pruritus in none, and the papain in all the subjects. It is concluded that bile salts can cause pruritus, the dihydroxy more than the trihydroxy salts.

1467 SCLEROSING CHOLANGITIS AND OTHER INTRA-ABDOMINAL FIBROSES. *(E.)* Thompson, B. W.; Read, R. C. (VA Hosp., Little Rock, Arkansas). *Am. J. Surg.* 128(6):777-781, 1974.

Case histories are presented for 12 patients (11 male, 1 female, 28-75 yr) who had been operated on for sclerosing cholangitis. All were icteric; the bilirubin levels varied from 2.0-43 ml/100 ml, and alkaline phosphatase levels ranged from 6.8-180 King-Armstrong U/100 ml, most being over 60.0 U/100 ml. The most common presenting symptoms were vague upper abdominal pain and intermittent but progressive jaundice extending up to 3 yr preceding hospitalization. Six of the patients had other diseases as well; 3 had stones in the gallbladder; 1 had ulcerative colitis; one had both retroperitoneal fibrosis and retractile mesenteritis; and 1 was hypersensitive to a drug. Large nodes along the common bile duct and nonspecific inflammation and fibrosis in the extrahepatic biliary tree were found. Operation was necessary to diagnose and decompress the biliary tract. Despite T tube drainage and antibiotic and steroid therapy, 8 of the patients have died. It is concluded that this disease is only one manifestation of a generalized fibrosis which is secondary to a hypersensitivity vasculitis.

1468 BILE DUCT ADENOMATOSIS OF THE LIVER: A MISLEADING FINDING ON SURGICAL EXPLORATION OF THE ABDOMEN. *(E.)* Levin, S. E.; Dail, D. H.; Saik, R. P. (VA Hosp., San Diego, Calif.). *Am. Surg.* 41(2):106-108, 1975.

A 62 year old man underwent laparotomy for duodenal obstruction. It was noticed that his liver was covered with tan to green-black subcapsular nodules. A biopsy and frozen section diagnosis were immediately performed. The growths were identified as bile duct adenomatosis, benign in nature, and hamartomatous in origin, and not metastases of a possible duodenal neoplasm, as suspected. The duodenal obstruction was found to be due to ulceration. It is suggested that this type of condition be confirmed by frozen section pathology to rule out possible coexistent malignancy.

1469 ANALYSIS OF GALLBLADDER BILE IN MORBID OBESITY. *(E.)* Freeman, J. B.; Meyer, P. D.; Printen, K. J.; Mason, E. E.; DenBesten, L. (Univ. VA Hosp., Univ. Iowa, Iowa City). *Am. J. Surg.* 129(2):163-166, 1975.

The chemical composition of gallbladder bile from morbidly obese patients was compared with that of normal controls to study gallstone etiology in morbid obesity. Bile samples were withdrawn from 11 patients, 100% in excess of ideal weight, during gastric bypass surgery. Samples were also taken from 11 controls undergoing abdominal surgery. All samples were immediately cultured and centrifuged. The bile from the obese patients fell outside the micellar zone (upper limit of cholesterol solubility) while that from the controls fell within the zone, with 1 exception. Cholesterol crystals were found from 4 of the obese patients and from none of the controls. In the obese patients the bile concentrations of cholesterol, phospholipids and bile salts were 26, 51.7 and 131 mM/liter resp., while the corresponding values in the controls were 15.0, 40.0, and 169.0 mM/liter. The cholesterol solubilizing capacity of the bile expressed as the lithogenic index was 6.3 in the obese and 14.8 in the control patients. It is concluded that since the bile of the obese patients was supersaturated with cholesterol, the previous correlations of obesity with gallstone formation are substantiated.

1470 EVALUATION OF THE RESULTS OF POSTOPERATIVE CHOLANGIOMANOMETRY PERFORMED DURING DRAINAGE. *(Rus.)* Porembskii, O. B.; Bronshtein, E. L. (S. M. Kirov Mil. Med. Acad., Leningrad, USSR). *Klin. Khir.* (7):9-13, 1973.

Expediency of a temporary bile drainage after biliary duct surgery was investigated. Studies included measurements of duct pressure which depends on the functional condition of Oddi's sphincter, secretory action of the liver and pancreas, and intestinal emptying. Seventy-six patients were studied after cholecystotomy; 34 also had choledochotomy. Surgery included temporary bile drainage through the stump of the gallbladder duct. Cholangiomanometry was first performed 6-8 hr after surgery, then each morning before the removal of the drain. The critical

pressure was close to normal in most cases. Maximum pressure (185 mm H_2O) was reached 4-6 days after surgery. On the 4th day, edema of duodenal papilla was reduced. Nitroglycerin contributed to greater flow capacity of the duodenal papilla accompanied by the onset of Oddi's sphincter dystonia. Comparison of cholangiographic data during drainage and cholangiomanometry performed 7-15 days after surgery indicated that following the restoration of permeability of the terminal part of the bile duct, bile excretion through the drain did not stop. Cholangiomanometry showed obstruction of the bile ducts while the contrast medium was retained in the duodenum for 3-5 min (normally, 30 sec). Impairment of duodenal emptying resulted in rising pressure which can account for the biliary hypertension after the restoration of Oddi's sphincter. Temporary bile drainage is recommended after surgery to prevent postoperative ductal hypertension.

1471 HEPATICOGASTROSTOMY: ULCEROGENIC PREPARATION OR THERAPEUTIC ALTERNATIVE. *(E.)* Warren, K. W.; Harmon, J. W. (Lahey Clin. Found., Boston, Mass.). *Ann. Surg.* 181(1):5-8, 1975.

Seven cases of hepaticogastrostomy for biliary obstruction were reviewed to determine whether the operation led to gastric ulceration. Three had been operated on for benign obstruction, 3 for malignant obstruction, and 1 for a congenital anomaly. Jaundice was relieved in 5 of the cases (71%); results were unsatisfactory in 1 and unknown in 1. Two of the patients developed upper gastrointestinal tract hemorrhage. One died, but the other recovered to live 2 yr and 4 months with no further hemorrhage. Four patients showed no signs of ulceration during follow-up periods ranging from 4 months to 21 yr. It is concluded that this operation has a high rate of success, despite theoretical considerations, and has definite merit as a bypass operation.

1472 GALLSTONE DISEASE. *(E.)* Coyne, M. J.; Schoenfield, L. J. (Cedars-Sinai Med. Cent., Los Angeles, Calif.). *Postgrad. Med.* 57(1): 153-162, 1975.

1473 PATHOGENESIS OF GALLSTONE FORMATION IN MAN: A REVIEW. *(E.)* Goswami, S. K.; Frey, C. F. (Dep. Surg., Univ. Michigan, Ann Arbor). *Univ. Michigan Med. Cent. J.* 40(1):32-37, 1974.

1474 CHOLELITHIASIS. *(Fl.)* Fevery, J. (St. Rafael Gen. Hosp., Louvain, Belgium). *Tijdschr. Geneesk.* 29(14):657-664, 1973.

1475 PATHOPHYSIOLOGY OF CHOLELITHIASIS. *(E.)* Redinger, R. N. (Univ. West. Ontario Fac. Med., London, Canada). *Schweiz. Med. Wochenschr.* 104(47):1673-1679, 1974.

1476 CHOLELITHIASIS AND ILEAL PATHOLOGY IN CHILDHOOD. *(E.)* Pellerin, D.; Bertin, P.; Nihoul-Fekete, C.; Ricour, C. (Necker Hosp. Sick Child., Paris, France). *J. Pediatr. Surg.* 10(1): 35-41, 1975.

1477 CHOLESTEROL GALLSTONES: PATHOGENESIS AND MEDICAL TREATMENT. *(Fr.)* Shaffer, E. A.; Beaudoin, M. (Montreal Gen. Hosp., Canada). *Union Med. Can.* 103(12):2061-2068, 1974.

1478 CHOLEDOCHOLITHIASIS: ADVANCES AND OUTLOOK FOR SURGICAL TREATMENT. *(Fr.)* Ryncki, P. V. (Dep. Surg., Univ. Lausanne, Switzerland). *Schweiz. Med. Wochenschr.* 104(47):1684-1694, 1974.

1479 VARIATION OF HEPATIC BILE COMPOSITION FOLLOWING CHOLECYSTECTOMY IN PATIENTS WITH PREVIOUS GALLSTONES. *(E.)* Boyer, J. L.; Bloomer, J. R.; Maddrey, W. C.; Tilson, D. (Yale Univ. Sch. Med., New Haven, Conn.). *Yale J. Biol. Med.* 47(4):211-217, 1974.

1480 DIAGNOSIS AND TREATMENT OF ENTEROBILIARY FISTULAS IN CHOLELITHIASIS. *(Rus.)* Panchenkov, R. T.; Lezhnev, A. N. (Moscow Inst. Med. Stomatol., USSR). *Khirurgiia (Mosk.)* (11):57-63, 1973.

1481 EXPERIMENTAL STUDIES ON FACTORS INITIATING PANCREATITIS ASSOCIATED WITH GALLSTONES, ESPECIALLY CHOLESTEROL STONES. *(Jpn.)* Yamazaki, H. (Kyoto Univ. Med. Sch., Japan). *Arch. Jpn. Chir.* 42(2):169-183, 1973.

1482 SPASM AND OPERATIVE CHOLANGIOGRAPHY. *(E.)* Chessick, K. C.; Black, S.; Hoye, S. J. (Tufts Univ. Sch. Med., Medford, Mass.). *Arch. Surg.* 110(1):53-57, 1975.

1483 INTRAOPERATIVE CHOLANGIOGRAPHY: CAUSES OF TECHNICAL ERRORS. *(Pol.)* Poltorak, J.; Wiechno, W.; Zakrzewski, M. (Bielanski Hosp., Warsaw, Poland). *Wiad. Lek.* 27(10):861-864, 1974.

1484 PERCUTANEOUS TRANSHEPATIC CHOLANGIOGRAPHY IN THE MANAGEMENT OF OBSTRUCTIVE JAUNDICE. *(E.)* Legge, D. A.; Hennessy, T. (St. Finbarr's Hosp., Cork, Ireland). *Ir. J. Med. Sci.* 144(1): 30-43, 1975.

1485 SOME PATHOGENETIC PRINCIPLES FOR SURGICAL TACTICS IN THE TREATMENT OF OBSTRUCTIVE JAUNDICE. *(Rus.)* Napalkov, P. N.; Vitlin, V. I. (Leningrad Med. Inst. Sanit. Hyg., USSR). *Khirurgiia (Mosk.)* (11):33-38, 1973.

1486 SKIN PROLYL HYDROXYLASE IN PATIENTS WITH OBSTRUCTIVE JAUNDICE. *(E.)* Than, T.; McGee, J. O'D.; Sokhi, G. S.; Patrick, R. S.; Blumgart, L. H. (R. Infirm., Glasgow, Scotland). *Lancet* 2(7884):807-808, 1974.

1487 CHOLEDOCHAL CYSTS. A REVIEW OF THE CYSTIC ANOMALIES OF THE BILIARY TREE. *(E.)* Olbourne, N. A. (Northwick Park Hosp., Harrow, England). *Ann. R. Coll. Surg. Engl.* 56(1):26-32, 1975.

1488 EXPERIENCE WITH CHOLEDOCAL [SIC!] CYST. *(E.)* Valayer, J.; Alagille, D. (St. Vincent de Paul Hosp., Paris, France). *J. Pediatr. Surg.* 10(1):65-68, 1975.

1489 WAYS TO DECREASE THE MORTALITY RATE IN ACUTE CHOLECYSTITIS IN ELDERLY PATIENTS. *(Rus.)* Struchkov, V. I.; Lokhvitskii, S. V.; Misnik, V. I. (I.M. Sechenov 1st Moscow Med. Inst., USSR). *Khirurgiia (Mosk.)* (11):39-44, 1973.

1490 ACUTE ACALCULOUS CHOLECYSTITIS. *(Fr.)* Favre, J. P.; Perrin, M.; David, M.; Piganiol, G.; Viard, H. (Bocage Hosp., Dijon, France). *Lyon Chir.* 70(5):307-309, 1974.

1491 ACUTE CHOLECYSTITIS IN CHILDREN. *(E.)* Pieretti, R.; Auldist, A. W.; Stephens, C. A. (Hosp. Sick Child., Toronto, Canada). *Surg. Gynecol. Obstet.* 140(1):16-18, 1975.

1492 A COMPARATIVE APPRAISAL OF EMPHYSEMATOUS CHOLECYSTITIS. *(E.)* Mentzer, Jr., R. M.; Golden, G. T.; Chandler, J. G.; Horsley, III, J. S. (Dep. Surg., Univ. Virginia, Charlottesville). *Am. J. Surg.* 129(1):10-15, 1975.

1493 THE SIGNIFICANCE OF ELECTROMANOMETRY TO ASSESS BENIGN DISEASES OF THE PAPILLA OF VATER. *(E.)* Boeckl, O.; Hell, E.; Laszcz, M.; Zimmermann, G. (Oberndorf Hosp., Salzburg, Austria). *Eur. Surg. Res.* 6(Suppl. 1):56-57, 1974.

1494 DIAGNOSIS AND TREATMENT OF CANCER OF THE PAPILLA OF VATER. *(Rus.)* Shkrob, O. S.; Lopata, Iu. M.; Safronov, V. V. (I.M. Sechenov 1st Moscow Med. Inst., USSR). *Khirurgiia (Mosk.)* (11):50-57, 1973.

1495 MALIGNANT NEOPLASMS OF THE GALLBLADDER. *(Rus.)* Dasaev, A. N.; Goriacheva, P. I.; Pushkin, L. N. (no affil.). *Khirurgiia (Mosk.)* (1):78-79, 1974.

1496 PRIMARY CARCINOMA OF THE GALLBLADDER. *(E.)* Prakash, A. T. M.; Sharma, L. K.; Pandit, P. N. (All India Inst. Med. Sci., New Delhi). *Br. J. Surg.* 62(1):33-36, 1975.

1497 INTRAHEPATIC BY-PASS OPERATIONS IN CARCINOMA OF THE BILIARY TRACT. *(Fr.)* Patel, J. C.; Chapuis, Y.; Delaitre, B.; Desvignes, G.; Paulian, B. (Cochin Hosp., Paris, France). *J. Chir. (Paris)* 107(5/6):521-536, 1974.

1498 BILIARY TRACT CARCINOMA INVOLVING THE AMPULLA OF VATER AND THE PANCREATIC DUCT TREATED BY RADICAL LEFT HEPATECTOMY AND DUODENO-PANCREATECTOMY IN ONE STAGE. *(Fr.)* Marchal, G.; Vernette, M.; Roustan, J.; Henry, G. (no affil.). *J. Chir. (Paris)* 107(5/6):555-578, 1974.

1499 AGENESIS OF THE GALLBLADDER: REPORT OF TWO CASES. *(E.)* Sachdeva, H. S.; Agrawal, B. K.; Mittal, V. K. (Postgrad. Inst. Med. Educ. Res., Chandigarh, India). *Am. Surg.* 41(2):109-110, 1975.

1500 TORSION OF THE GALLBLADDER. *(E.)* Neher, R. G.; Warner, G. W. (St. Benedict's Hosp., Jerome, Idaho). *Abdom. Surg.* 17(2):32-35, 1975.

1501 BILE LAKES IN CONGENITAL BILIARY ATRESIA. *(E.)* Fonkalsrud, E. W.; Arima, E. (Univ. California Sch. Med., Los Angeles). *Surgery* 77(3):384-390, 1975.

1502 VARIATIONS AND ANOMALIES OF THE PAPILLA OF VATER, THE PANCREAS AND THE BILIARY DUCT SYSTEM. *(E.)* Phillip, J.; Koch, H.; Classen, M. (Dep. Med., Univ. Erlangen-Nuremberg, Erlangen, Germany). *Endoscopy* 6(2):70-77, 1974.

1503 A CASE OF ANOMALOUS BILE DUCT ASSOCIATED WITH CHOLELITHIASIS. *(E.)* Nagase, M.; Kido, S.; Detoyama, M.; Fujimura, M. (Yamato-Takada City Hosp., Japan). *Arch. Jpn. Chir.* 42(4):377-380, 1973.

1504 CHOLECYSTOHEPATIC SERENDIPITY. *(E.)* Fentiman, I. S.; Ramsay, G. S. (R. London Homoeopath. Hosp., England). *Br. J. Clin. Pract.* 29(1):21-22, 1975.

1505 PAIN REFERABLE TO THE BILIARY TRACT AND HYPERPLASIA OF THE GALLBLADDER MUCOSA. *(Fr.)* Jekic, M. (Zemun Clin. Hosp., Belgrade, Yugoslavia). *Helv. Chir. Acta* 41(5/6):825-828, 1974.

1506 AN ABERRANT RIGHT HEPATIC DUCT DISCHARGING INTO THE NECK OF THE GALLBLADDER. *(It.)* Fedele, E. (Nuovo Regina Margherita Hosp., Rome, Italy). *Chir. Gastroenterol.* 8(1):104-113, 1974.

1507 RADIOLOGICAL DIAGNOSIS OF HYPERPLASTIC GALLBLADDER DISEASE. *(Ger.)* Peter, M.; Vargha, G.; Fedor, E.; Bekesi, L. (Med. Univ., Debrecew, Hungary). *Radiol. Diagn. (Berl.)* 14(5):567-574, 1973.

1508 DIFFERENTIATION BETWEEN LABILE AND PERSISTENT HYPERTENSION OF THE SPHINCTER OF ODDI. *(Fr.)* Clemens, M. (Karcag Hosp., Hungary). *Lyon Chir.* 70(4):232-233, 1974.

1509 EVALUATION OF X-RAY CONTRAST AND INSTRUMENTAL INVESTIGATIONS IN BILIARY TRACT SURGERY. *(Rus.)* Belichenko, I. A.; Obukhov, V. A.; Gorbunov, V. V. (Moscow Inst. Med. Stomatol., USSR). *Khirurgiia (Mosk.)* (11):63-67, 1973.

1510 ENTEROBILIARY ANASTOMOSES IN BILIARY TRACT SURGERY. *(Rus.)* Kalchenko, I. I.; Lys, P. V.; Khokholia, V. P. (Kiev Inst. Postgrad. Med., USSR). *Khirurgiia (Mosk.)* (11):44-50, 1973.

1511 ENTEROBILIARY ANASTOMOSES. *(Ger.)* Haas, D.; Vogt, B. (Canton. Hosp., Lucerne, Switzerland). *Helv. Chir. Acta* 41(5/6):819-821, 1974.

1512 ERRORS AND COMPLICATIONS IN SURGERY OF THE GALLBLADDER EXTRAHEPATIC BILIARY TRACT. *(Rus.)* Shuvayeva, V. I. (M. F. Vladimirskii MONIKI, Moscow, USSR). *Khirurgiia (Mosk.)* (11):121-126, 1973.

1513 TRANSDUODENAL PAPILLOTOMY: SURGICAL TECHNIQUE, INDICATIONS, AND RESULTS. *(Ger.)* Willenegger, H.; Kaiser, C.; Roth, B.; Muller, J. (Liestal Canton. Hosp., Switzerland). *Helv. Chir. Acta* 41(5/6):808-813, 1974.

1514 PAPILLOTOMY USING THE SOLER-ROIG CATHETER. *(Ger.)* Thalmann, H.; Roth, H. (Canton. Hosp., Munsterlingen, Switzerland). *Helv. Chir. Acta* 41(5/6):823-824, 1974.

1515 EARLY COMPLICATIONS AFTER PAPILLOTOMY. *(Pol.)* Wladzinski, J. (Mil. Med. Acad., Lodz, Poland). *Wiad. Lek.* 27(7):619-623, 1974.

1516 INDICATIONS FOR AND RESULTS FROM THE ROUX-EN-Y PROCEDURE USING A LONG JEJUNAL LOOP FOR THE COMMON BILE DUCT. *(Ger.)* Schweingruber, B.; Gemsenjager, E.; Meier, A. L. (St. Clara Hosp., Basel, Switzerland). *Helv. Chir. Acta* 41(5/6):815-817, 1974.

1517 CHOLEDOCHODUODENOSTOMY AND SPHINCTEROPLASTY. *(Afr.)* Fichardt, J. B.; De Villiers, D. R.; Bornman, P. C. (Dep. Surg., Orange Free State Univ., Bloemfontein, South Africa). *S. Afr. Med. J.* 48(59): 2457-2459, 1974.

1518 REFLUX OF THE DUODENAL CONTENTS INTO THE BILE DUCTS FOLLOWING CHOLEDOCHODUODENOSTOMY. *(Pol.)* Kondrak, R.; Syc, S. (Prov. Hosp., Opole, Poland). *Wiad. Lek.* 27(5):435-438, 1974.

1519 PROGRESS IN BILIARY STRICTURE REPAIR. *(E.)* Braasch, J. W.; Warren, K. W.; Blevins, P. K. (Lahey Clin. Found., Boston, Mass.). *Am. J. Surg.* 129(1):34-37, 1975.

1520 PLASTIC REPAIR OF THE COMMON BILE DUCT BY A PEDUNCULATED FRAGMENT OF THE GALLBLADDER. *(Fr.)* Sandblom, P.; Tabrizian, M. (Dep. Surg. B., Univ. Lausanne, Switzerland). *Helv. Chir. Acta* 41(5/6):789-792, 1974.

1521 PROBLEMS OF INTRAOPERATIVE FUNCTIONAL DIAGNOSIS OF SPHINCTER ODDI. *(E.)* Lynen, F. K. (Med. Fac., Rhenish-Westphal. Tech. Univ., Aachen, Germany). *Eur. Surg. Res.* 6(Suppl. 1):54-55, 1974.

1522 THE BIOLOGICAL CHARACTERISTICS OF STAPHYLOCOCCI ISOLATED FROM BILE DURING CHRONIC DISEASES OF THE GALLBLADDER AND BILE DUCTS. *(Rus.)* Orzhonikidze, Z. V.; Kurashvili, V. E.; Salakaia, R. G. (Tbilisi State Inst. Postgrad. Med., USSR). *Soobshch. Akad. Nauk Gruzinsk. SSR* 76(2):465-468, 1974.

1523 THE EFFECT OF PENTAZOCINE ON THE FUNCTION OF THE SPHINCTER OF AMPULLA OF VATER. *(Pol.)* Zakrzewski, M.; Poltorak, J. L.; Ruka, W. (Bielanski Hosp., Warsaw, Poland). *Pol. Tyg. Lek.* 29(14):585-586, 1974.

See also, 0926, 1093, 1136, 1138, 1147, 1148, 1168, 1174, 1191, 1202, 1203, 1204, 1205, 1206, 1207, 1208, 1209, 1210, 1240, 1283, 1287, 1328, 1329, 1357, 1403, 1422, 1445, 1548, 1556.

GENERAL

1524 CALCIUM INFUSION TEST BEFORE AND AFTER TOTAL GASTRECTOMY IN THE ZOLLINGER-ELLISON SYNDROME. *(E.)* Morrow, D. J; Passaro, Jr., E. (VA Wadsworth Hosp. Cent., Los Angeles, Calif.). *Am. J. Surg.* 129(1):62-66, 1975.

Data were obtained on calcium infusion studies in Zollinger-Ellison patients before and after total gastrectomy to further document the value of the test in establishing the diagnosis in the absence of significant hypergastrinemia. Prior to total

gastrectomy, 14 patients with Zollinger-Ellison syndrome underwent calcium gluconate infusion at the rate of 5 mg of elemental calcium/kg/hr over a 3 hr period. Eleven Zollinger-Ellison patients were studied with a calcium infusion after total gastrectomy. Before total gastrectomy, the basal serum gastrin levels ranged from 100 to 6000 pg/ml. The basal acid secretion ranged from 4.5 to 34 mEq/hr. Calcium infusion produced a mean increase in serum gastrin concentration of 106% and a mean increase in acid secretion of 280%. Maximal serum gastrin increases over basal were 41 to 900% after calcium infusion. The 11 patients who had total gastrectomy were able to be subdivided into 3 groups based on their basal serum gastrin levels and their responses to calcium infusion. In the first group, 4 patients demonstrated an increase in serum gastrin concentration of 100% or more. At least 3 of these 4 had residual tumor after total gastrectomy. These same 3 patients had basal postoperative serum gastrin levels below 250 pg/ml. In the second group were 4 patients whose basal serum gastrin levels were elevated and not altered by induced hypercalcemia. Two of these died of the disease, both with extensive hepatic metastases at autopsy. A third is doing well 8 yr later, despite known pancreatic tumor and extensive hepatic metastases. The 4th is well, 6 yr after operation. In the third group were 3 patients each having a low basal serum gastrin concentration which did not increase with calcium infusion. Either no tumor was found or all known tumor was removed in these patients. It is concluded that in a preoperative group of patients the serum gastrin and gastric acid response to calcium infusion may be highly diagnostic. After total gastrectomy, the gastrin response to such infusion correlates well with presence or absence of tumor.

1525 LONG-TERM EVALUATION OF THE CELIAC BAND SYNDROME. *(E.)* Evans, W. E. (Ohio State Univ. Hosp., Columbus). *Surgery* 76(6):867-871, 1974.

Fifty-nine patients (49 women and 10 men, aged 13-68 yr) with celiac compression syndrome were observed for 3 to 11 yr following their initial diagnoses. Operations were performed on 47 of these patients; the other 12 were treated nonsurgically. Operation consisted of division of the median arcuate ligament and clearing of all tissue overlying the celiac axis. Over 70% of these patients had had a previous abdominal operation without relief of their symptoms and all had long histories of abdominal complaints. Of the 47 who underwent celiac band release, 3 had cholecystectomy simultaneously. Additional subsequent abdominal procedures have been performed on 13 patients. During the first 6 months after surgery, 39 patients (83% of those undergoing only celiac band release) were asymptomatic, while 8 (17%) were not improved. At the end of the follow up period, only 41% remained symptom free. Fifty percent were not benefited at all. Eight of these underwent additional surgery and are now symptom free. Of the 12 patients treated nonsurgically, 9 (75%) are asymptomatic, 1 is improved, and only 2 continue to be symptomatic. Of the pain free group, 4 responded to psychiatric treatment, and 3 became symptom free when taken off milk products. There was no correlation between symptoms and the results of either surgical or nonsurgical treatment. These results suggest that compression of the celiac artery cannot likely be implicated as an identifiable syndrome causing abdominal pain. A skeptical and cautious approach to these patients is suggested.

1526 INTESTINAL MANIFESTATIONS OF FABRY'S DISEASE. *(E.)* Rowe, J. W.; Gilliam, J. I.; Warthin, T. A. (West Roxbury VA Hosp., Mass.). *Ann. Intern. Med.* 81(5):628-631, 1974.

The radiographic and clinical manifestations of intestinal involvement in 4 patients (all men, aged 31-50 yr) with Fabry's disease are presented. Histologic studies in 2 of these cases revealed normal small and large intestinal mucosa with accumulation of glycolipid in autonomic ganglia, neurons of myenteric plexuses, and smooth muscle cells and blood vessels. Episodic diarrhea was a prominent complaint in 3 of these 4 patients. In 1 patient, diarrhea was the only clinical manifestation. These attacks were unpredictable in onset or duration, progressive in frequency and severity, and were often associated with crampy abdominal pain, and remitted spontaneously. The stools were loose, free of blood, of normal color, and were not buoyant or foul smelling. Results of sigmoidoscopy were normal. There was no strong clinical or chemical evidence of malabsorption in any patient, although 1 was studied while his bowel symptoms were in remission. Radiologic studies revealed similar changes in the small and large bowels of all 4 patients. Small bowel changes consisted of thickening of folds and mild dilatation throughout and the presence of an unusual granular appearance of the ileum. Barium enemas showed loss of the colonic haustral markings, particularly in the more distal colonic segments. Serial studies on the symptomatic patients, before and after the onset of diarrhea, showed progression of the radiographic changes. There was no evidence of malabsorption or gastrointestinal blood loss in any patient. It is suggested that intestinal involvement, either silent or clinically evident, may be more common in Fabry's disease than has been previously recognized.

1527 ULTRASTRUCTURAL STUDIES OF THE PAROTID GLANDS IN PAROTID SWELLING. *(Ger.)* Donath, K.; Seifert, G. (Inst. Pathol., Univ. Hamburg, Germany). *Virchows Arch.* [*Pathol. Anat.*] 365(2):119-135, 1975.

1528 END RESULTS OF SURGICAL TREATMENT OF 622 TUMORS OF THE SALIVARY GLANDS. *(It.)* Catania, C. V.; Galante, E.; Bandieramonte, G.; Salvadori, B. (Natl. Inst. Cancer Res. Ther., Milan, Italy). *Tumori* 60:307-316, 1974.

1529 TUMORS OF SALIVARY GLANDS. AN ANALYSIS OF 752 CASES. *(E.)* Richardson, G. S.; Dickason, W. L.; Gaisford, J. C.; Hanna, D. C. (West. Pennsylvania Hosp., Pittsburgh). *Plast. Reconstr. Surg.* 55(2):131-138, 1975.

1530 SERIAL MEASUREMENTS OF VITAMIN B-12 AND VITAMIN B-12-BINDING CAPACITY IN MARASMIC KWASHIORKOR. *(E.)* Lejeune-Lenain, C.; Fondu, P. (Univ. Brussels Cent. Afr. Cent. Sci. Med. Stud., Lwiro, Zaire). *Clin. Chim. Acta* 59(1):81-86, 1975.

1531 EMERGENCY TREATMENT OF LARGE NUMBERS OF CHILDREN WITH SEVERE PROTEIN-CALORIE MALNUTRITION. *(E.)* Ifekwunigwe, A. E. (Cent. Health Sci., Univ. California, Los Angeles). *Am. J. Clin. Nutr.* 28(1):79-83, 1975.

1532 BIOCHEMICAL DETECTION OF THIAMIN DEFICIENCY IN INFANTS AND CHILDREN IN THAILAND. *(E.)* Pongpanich, B.; Srikrikkrich, N.; Dhanamitta, S.; Valyasevi, A. (Ramathibodi Hosp., Mahidol Univ., Bangkok, Thailand). *Am. J. Clin. Nutr.* 27(12):1399-1402, 1974.

1533 PSYCHOSOMATIC ASPECTS OF GASTROINTESTINAL DISEASE. *(E.)* McKegney, F. P. (Univ. Vermont Coll. Med., Burlington). *Postgrad. Med.* 57(1):43-54, 1975.

1534 FISTULAS IN THE AREAS OF THE ESOPHAGUS, STOMACH, AND BILE DUCTS. *(Ger.)* Schriefers, K. H.; Gok, Y. (Munic. Hosp., Loblenz, Germany). *Langenbecks Arch. Chir.* 337:119-125, 1974.

1535 LONG-TERM RESULTS OF RECONSTRUCTIVE GASTROJEJUNODUODENOPLASTY. *(Rus.)* Chistova, M. A.; Chistov, L. V. (I. M. Sechenov 1st Moscow Med. Inst., USSR). *Khirurgiia (Mosk.)* (2):34-42, 1974.

1536 POSTOPERATIVE PARESIS OF THE STOMACH AND SMALL INTESTINE. *(Rus.)* Makhov, N. I.; Seleznev, G. F. (V.A. Ostroumov Munic. Clin. Hosp. No. 33, Moscow, USSR). *Khirurgiia (Mosk.)* (11):5-11, 1973.

1537 CLINICAL TRIAL OF A NEW PERISTALTIS-PRODUCING DRUG ACTING ON GASTROINTESTINAL SYNAPSES. *(Fr.)* Cinqualbre, J.; Lampert, M.; Avalos, S.; Forster, E. (no affil.). *J. Med. Strasbourg* 4(12):973-976, 1973.

1538 GASTROINTESTINAL AND HEPATIC COMPLICATIONS AFFECTING PATIENTS WITH RENAL ALLOGRAFTS. *(E.)* Aldrete, J. S.; Sterling, W. A.; Hathaway, B. M.; Morgan, J. M.; Diethelm, A. G. (Univ. Alabama Sch. Med., Birmingham). *Am. J. Surg.* 129(2): 115-124, 1975.

1539 PRECANCEROUS LESIONS IN THE GASTROINTESTINAL TRACT. *(Ger.)* Eder, M. (Pathol. Inst., Univ. Munich, Germany). *Langenbecks Arch. Chir.* 337:758-761, 1974.

1540 VALUE OF X-RAY EXAMINATION IN THE EVALUATION OF PRECANCER IN THE GASTROINTESTINAL TRACT. *(Ger.)* Frik, W. (Med. Fac., Rhenish-Westphal. Tech. Coll., Aachen, Germany). *Langenbecks Arch. Chir.* 337:764-765, 1974.

1541 HEREDITARY POLYPOID DISEASES OF THE GASTROINTESTINAL TRACT. A WORKING CLASSIFICATION. *(E.)* Sachatello, C. R.; Griffen, Jr., W. O. (Univ. Kentucky Med. Cent., Lexington). *Am. J. Surg.* 129(2):198-203, 1975.

1542 THE ZOLLINGER-ELLISON SYNDROME. CASE REPORT. *(E.)* Pannall, P.; Van Den Berg, W. P. J. (Bloemfontein Acad. Hosp., South Africa). *S. Afr. Med. J.* 48(59):2489-2491, 1974.

1543 CARCINOMA OF THE THYROID MEDULLA ASSOCIATED WITH BILATERAL PHEOCHROMOCYTOMA: SIPPLE'S SYNDROME WITH A PROTRACTED COURSE. *(Fr.)* Viguie, R.; Mihaud, G.; Vesin, P.; Dervichian, M.; Gardies, A.; Cohen, A.; Ancenay, M. (Hosp. Cent., Argenteuil, France). *Arch. Fr. Mal. App. Dig.* 63(5):395-400, 1974.

1544 NON-HODGKIN'S MALIGNANT LYMPHOMAS OF UPPER GASTROINTESTINAL AND RESPIRATORY TRACTS: NATURAL HISTORY AND RESULTS OF RADIOTHERAPY. *(Fr.)* Brugere, J.; Schlienger, M.; Gerard-Marchant, R.; Tubiana, M.; Pouillart, P.; Cachin, Y. (Gustave Roussy Inst., Villejuif, France). *Bull. Cancer (Paris)* 61(1): 79-92, 1974.

1545 SURGICAL TREATMENT OF PATIENTS WITH BLEEDING FROM VARICES OF THE ESOPHAGUS AND STOMACH. *(Rus.)* Patsiora, M. D.; Tsatsanidi, K. N.; Sherzinger, A. G.; Semenov, V. S. (I. M. Sechenov 1st Moscow Med. Inst., USSR). *Khirurgiia (Mosk.)* (2):3-8, 1974.

1546 ANALYSIS OF DEATHS IN ACUTE GASTRODUODENAL BLEEDING. *(Rus.)* Briskin, B. S. (Moscow Inst. Med. Stomatol., USSR). *Khirurgiia (Mosk.)* (2):8-14, 1974.

1547 BLEEDING IN THE SMALL AND LARGE INTESTINE AND THE RECTUM IN INFANCY AND CHILDHOOD. *(Ger.)* Hasse, W. (Rudolf Virchow Munic. Hosp., Berlin, Germany). *Langenbecks Arch. Chir.* 337: 594-595, 1974.

1548 HEMOBILIA AS THE CAUSE OF SERIOUS HEMATEMESIS. REPORT OF A CASE. *(E.)* Bjorneby, S. (Sarpsborg Hosp., Norway). *Acta Chir. Scand.* 140(7):576-577, 1974.

1549 CONTRIBUTION OF THE K88 ANTIGEN OF Escherichia coli TO ENTEROPATHOGENICITY; PROTECTION AGAINST DISEASE BY NEUTRALIZING THE ADHESIVE PROPERTIES OF K88 ANTIGEN. *(E.)* Jones, G. W.; Rutter, J. M. (Inst. Res. Anim. Dis., Compton, England). *Am. J. Clin. Nutr.* 27(12):1441-1449, 1974.

1550 DIFFERENT CLASSES OF IMMUNOGLOBULINS IN THE INTESTINAL TRACT OF MICE AFTER ORAL E. coli INFECTION. *(Rus.)* Dushkin, V. A.; Podoprigora, G. I.; Intizarov, M. M. (Sci. Res. Lab. Exp. Biol. Models, Moscow Region, USSR). *Biull. Eksp. Biol. Med.* 77(11):74-76, 1974.

1551 A STUDY OF THE BIOLOGICAL PROPERTIES OF E. coli STRAINS AND ISOLATED COLI BACTERIOPHAGES. *(Rus.)* Kuchashvili, K. I. (Tbilisi Sci. Res. Inst. Vaccines Sera, USSR). *Soobshch. Akad. Nauk Gruzinsk. SSR* 76(2):477-480, 1974.

1552 SHIGELLOSIS IN DAY-CARE CENTRES. *(E.)* Weissman, J. B.; Schmerler, A.; Gangarosa, E. J.; Marier, R. L.; Lewis, J. N. (Cent. Dis. Control, Atlanta, Georgia). *Lancet* 1(7898):88-90, 1975.

1553 CHOLERA IN AFRICA: A MESSAGE FOR THE WEST. *(E.)* Goodgame, R. W.; Greenough, III, W. B. (Dep. Med. John Hopkins Univ., Baltimore, Md.). *Ann. Intern. Med.* 82(1):101-106, 1975.

1554 EFFECT OF REVACCINATION IN PERSONS IMMUNIZED INITIALLY WITH WHOLE-CELL AND TOXOID CHOLERA VACCINES. *(Rus.)* Kiseleva, I. E.; Domaradksii, I. V.; Erokhin, E. P.; Kirdeev, V. K.; Kiseleva, V. I.; Rassudov, S. M.; Taranova, V. N.; Turchinov, G. A.; Shoshiev, L. N. (Rostov-on-Don Antiplague Sci. Res. Inst., USSR). *Zh. Mikrobiol. Epidemiol. Immunobiol.* (6):64-67, 1974.

1555 MANAGEMENT OF ILEAL PERFORATION DUE TO TYPHOID FEVER. *(E.)* Kim, J. P.; Oh, S. K.; Jarrett, F. (Seoul Natl. Univ. Hosp., Korea). *Ann. Surg.* 181(1):88-91, 1975.

1556 Salmonella heidelberg INFECTION IN CAROLI'S SYNDROME. *(E.)* Waldram, R.; Vahrman, J.; Williams, R. (King's Coll. Hosp., London, England). *Gastroenterology* 68(1):151-153, 1975.

1557 ASSAY, CHARACTERIZATION, AND LOCALIZATION OF AN ENTEROTOXIN PRODUCED BY Salmonella. *(E.)* Koupal, L. R.; Deibel, R. H. (Food Res. Inst., Univ. Wisconsin, Madison). *Infect. Immun.* 11(1):14-22, 1975.

1558 SALMONELLOSIS AND ITS CONTROL. *(Rus.)* Birkovskii, Iu. E. (Kiev Sci. Res. Inst. Epidemiol. Microbiol. Parasitol., USSR). *Vrach. Delo* (10):144-147, 1974.

1559 DURATION OF INFECTION AND DISTRIBUTION OF Salmonella IN TISSUES OF EXPERIMENTAL ANIMALS. *(Rus.)* Sagitova, L. Ia.; Suptel, E. A. (Kiev Sci. Res. Inst. Infect. Dis., USSR). *Vrach. Delo.* (10):147-150, 1974.

1560 DEFECT IN RESISTANCE TO Salmonella typhimurium IN IRON-DEFICIENT RATS. *(E.)* Baggs, R. B.; Miller, S. A. (Dep. Nutr., Food Sci., Massachusetts Inst. Tech., Cambridge). *J. Infect. Dis.* 130(4):409-411, 1974.

1561 PROTECTION OF MICE BY ORAL IMMUNIZATION WITH A PATHOGENIC STRAIN OF HEAT-KILLED Salmonella typhimurium. *(Fr.)* Ivanoff, B.; Creach, O.; Fontanges, R. (Res. Cent., Army Health Dep., Lyon, France). *C. R. Soc. Biol. (Paris)* 168(4/5): 541-547, 1974.

1562 SPECIFIC IMMUNE RESPONSE AND IMMUNOGLOBULIN PRODUCTION IN INTESTINAL INFECTIONS. I. ANTIBODIES AND IMMUNOGLOBULINS IN THE SERUM OF PATIENTS SUFFERING FROM DYSENTERY AND SALMONELLOSIS. *(Rus.)* Golderman, S. Ia.; Sukhoroslova, L. I.; Starshinova, V. S.; Malchikovskii, R. G. (Moscow Sci. Res. Inst. Epidemiol. Microbiol., USSR). *Zh. Mikrobiol. Epidemiol. Immunobiol.* (6):55-59, 1974.

1563 ORGAN CULTURE OF HUMAN EMBRYONIC INTESTINE AS A MODEL FOR THE STUDY OF INTERACTIONS BETWEEN DYSENTERY BACTERIA AND CELLS. *(Rus.)* Maksimovich, N. A.; Radolitskaia, L. S.; Suptel, E. A. (Kiev Sci. Res. Inst. Infect. Dis., USSR). *Arkh. Patol.* 36(10):59-64, 1974.

1564 DIARRHEA. PATHOGENESIS AND DIAGNOSTIC TECHNIQUES. *(E.)* Phillips, S. F. (Mayo Clin., Rochester, Minn.). *Postgrad. Med.* 57(1): 65-76, 1975.

1565 CHOLEDOCHOCOLONIC FISTULA: AN UNUSUAL CAUSE OF CHRONIC DIARRHEA WITH MALABSORPTION. *(Fr.)* Modigliani, R.; Hautefeuille, P.; Matuchansky, C.; Parienti, C.; Hirsch, J. F.; Bernier, J. J. (St. Lazare Hosp., Paris, France). *Arch. Fr. Mal. App. Dig.* 63(6):503-508, 1974.

1566 THE ROLE OF CERTAIN GRAM-NEGATIVE BACTERIA FROM THE FAMILY ENTEROBACTERIACEAE IN THE ETIOLOGY OF DIARRHEA IN INFANTS. *(Pol.)* Kulak, M. (Oswiecim Munic. Hosp., Poland). *Wiad. Lek.* 27(6): 507-510, 1974.

1567 ACTIVITY OF CEFAZOLIN AGAINST DENSE POPULATIONS OF ENTEROBACTERIA. *(E.)* Greenwood, D.; Chan-Teoh, C. H.; O'Grady, F. (St. Bartholomew's Hosp., London, England). *Antimicrob. Agents Chemother.* 7(2):191-195, 1975.

1568 LACTOBACILLUS FLORA OF INTESTINE AND THE EFFECT OF ANTIBIOTICS ON IT. *(Rus.)* Privalova, R. I. (Sverdlovsk Med. Inst., USSR). *Antobiotiki* 19(2):172-176, 1974.

1569 MIXED INTESTINAL INFECTIONS. *(Rus.)* Roenko, S. I.; Reznik, M. M.; Borina, M. Ia., Sofienko, N. Ia.; Kulichkina, E. I. (5th United Hosp., Kiev, USSR). *Vrach. Delo* (12):120-123, 1974.

1570 USE OF A ONE-STEP COMPLEX ANGIOGRAPHIC METHOD IN DIAGNOSIS OF THE DISEASES OF ORGANS IN THE ABDOMINAL CAVITY. *(Rus.)* Portnoi, L. M.; Obmornov, L. E.; Shumskii, V. I.; Ender, L. A.; Stranin, V. G. (M.F. Vladimirskii MONIKI, Moscow, USSR). *Khirurgiia (Mosk.)* (11):16-22, 1973.

1571 MICROSCOPIC INTESTINAL FUNGI AND THEIR ANTIBACTERIAL ACTIVITY. *(Rus.)* Shevchenko, T. F. (Semipalatinsk Med. Inst., USSR). *Zh. Mikrobiol. Epidemiol. Immunobiol.* (6):92-94, 1974.

1572 MORPHOLOGICAL ALTERATIONS IN THE MICROVILLOUS BORDER OF VILLOUS EPITHELIAL CELLS PRODUCED BY INTESTINAL MICROORGANISMS. *(E.)* Erlandsen, S. L.; Chase, D. G. (Coll. Med., Univ. Iowa, Iowa City). *Am. J. Clin. Nutr.* 27(11):1277-1286, 1974.

1573 IS CHLORAMPHENICOL THE DRUG OF CHOICE FOR THE TREATMENT OF ENTERIC FEVER? COMPARATIVE STUDIES ON THE EFFECTS OF CHLORAMPHENICOL, TRIMETHOPRIM-SULPHAMETOXAZOLE AND β-LACTAM ANTIBIOTICS. *(E.)* Jonsson, M. (Inst. Infect. Dis., Karolinska Inst., Stockholm, Sweden). *Infection* 2(3):145-151, 1974.

1574 ENTERIC FEVER IN YOUNG YORUBA CHILDREN. *(E.)* Duggan, M. B.; Beyer, L. (Wesley Guild Hosp. Ilesha, Nigeria). *Arch. Dis. Child.* 50(1):67-71, 1975.

1575 EPIDEMIC VIRAL ENTERITIS IN A LONG-STAY CHILDREN'S WARD. *(E.)* Flewett, T. H.; Bryden, A. S.; Davies, H. (East Birmingham Hosp., England). *Lancet* 1(7897):4-5, 1975.

1576 ULCERATIVE NECROTIZING ENTERITIS IN PREMATURE INFANTS. STUDY OF 17 CASES. *(Fr.)* Mourot, M.; Beau, A.; Raubier, G.; Vert, P. (Univ. Hosp. Cent., Nancy, France). *Ann. Chir. Infant.* 15(2):119-134, 1974.

1577 DISORDERS IN MEMBRANE DIGESTION IN THE SMALL INTESTINE IN CHRONIC ENTEROCOLITIS. *(Rus.)* Frolkis, A. V.; Goranskaia, S. V.; Fedotova, O. I. (Dep. Prelim. Instr. Intern. Med., Petrozavodsk Univ., USSR). *Sov. Med.* (12):92-95, 1973.

1578 ENTEROCOLITIS IN ACUTE VIRAL RESPIRATORY INFECTIONS IN CHILDREN. *(Rus.)* Tsinserling, A. V.; Chastina, G. V. (Leningrad Inst. Pediatr. Med., USSR). *Arkh. Patol.* 36(10):54-59, 1974.

1579 INVESTIGATION OF MUCOPROTEINS IN CHRONIC ENTEROCOLITIS. *(Rus.)* Silchenko, T. S.; Vashchenko, G. S. (Dnepropetrovsk Sci. Res. Inst. Gastroenterol., USSR). *Vrach. Delo* (10):35-38, 1974.

1580 THERAPEUTIC EFFICIENCY OF METHYLMETHIONINE SULFONIUM CHLORIDE (VITAMIN U) IN PATIENTS WITH CHRONIC ENTEROCOLITIS. *(Rus.)* Trusov, V. V.; Vakhrushev, Ia. M. (Izhevsk Med. Inst., USSR). *Vrach. Delo* (12):78-82, 1974.

See also, 1132, 1134, 1151, 1152, 1155, 1167, 1178, 1214, 1217, 1289, 1426, 1522.

GENERAL

Parasitology

1581 LOCALIZATION OF THE CIRCULATING ANTIGEN WITHIN THE GUT OF Schistosoma mansoni. *(E.)* Nash, T. E. (Natl. Inst. Allergy Infect. Dis., Bethesda, Md.). *Am. J. Trop. Med. Hyg.* 23(6):1085-1087, 1974.

The indirect immunofluorescent technique was used to determine the location of the antigen in adult *Schistosoma mansoni* which appears in the circulation of mice and hamsters heavily infected with *S. mansoni* or *S. japonicum*. Antisera to the circulating antigen was raised by immunizing rabbits with a trichloroacetic acid soluble-chloroform insoluble fraction of adult *S. mansoni* coupled to methylated bovine serum albumin. Sections of adult *S. mansoni* and of infected mouse liver were incubated with this monospecific antiserum for 30 min at room temperature, followed by treatment with a 1:10 dilution of fluorescein conjugated goat anti-rabbit immunoglobulin G. Controls included slides treated with only normal rabbit serum, only conjugate, or neither normal rabbit serum or conjugate. Specific immunofluorescent

staining was not observed in any of the controls. However, with antiserum the border of the intestinal lining cells of the cecum with the lumen fluoresced very brightly, the lining cells themselves fluoresced to a lesser degree and the gut contents fluoresced occasionally. This corresponds to an area of diastase-fast periodic acid-Schiff positive staining, which confirms previous data indicating that this antigen is a polysaccharide. The antigen was not found elsewhere in the worms or in the liver of infected mice. These results indicate that the circulating antigen is localized to the lining cells of the cecum in male and female schistosomes, but as yet no physiological function can be ascribed to it.

1582 ANTIBODY-DEPENDENT CELL-MEDIATED DAMAGE TO SCHISTOSOMULA IN VITRO. *(E.)* Butterworth, A. E.; Sturrock, R. F.; Houba, V.; Rees, P. H. (Wellcome Trust Res. Labs., Nairobi, Kenya). *Nature* 252(5483):503-505, 1974.

The ^{51}Cr-release technique was adapted to assay damage to schistosomula of *Schistosoma mansoni in vitro* and used to provide evidence that normal human peripheral blood leukocytes, in the presence of sera from patients infected with *S. mansoni*, can damage labeled schistosomula in the absence of added complement. After overnight storage of schistosomula, the organisms were labeled with ^{51}Cr-sodium chromate by incubation of 10^4 organisms with 0.5-1 mCi of ^{51}Cr in a volume of less than 0.5 ml for 3-4 hr at 37 C. This procedure gave count rates of 50-300 c.p.s./100 organisms. Serum samples were obtained from patients with a long history of presumptive exposure to *S. mansoni* and from laboratory workers with no known exposure. The cytotoxicity test was carried out in a total volume of 0.3 ml in 38 x 7 mm sterile plastic tubes. At the end of the incubation period, 0.15 ml of the supernatant was removed, and both cell and supernatant tubes counted for ^{51}Cr. Cells alone, at a multiplicity of 2,000:1 to the target organisms, caused a slight increase in isotope release. Cells in combination with antibody caused a marked increase in isotope release which was significantly different from the effect of cells alone from 2 hr of incubation. By 19 hr, isotope release in the presence of cells and antibody was beginning to show a plateau effect. A total of 11 test sera and 4 control sera were examined. None of the sera was toxic for schistosomula in the absence of added leukocytes. These results indicate that schistosomula can be damaged *in vitro* by normal human peripheral blood leukocytes together with sera from infected patients but not with sera from control subjects. The effect is complement-independent in that heat-inactivated sera were used throughout. Preliminary observations indicate that maximum cytotoxic activity is associated with an eosinophil-rich polymorphonuclear leukocyte fraction.

1583 SCHISTOSOMIASIS IN NEW YORK CITY. *(E.)* Welton, J.; Imperato, P. J. (Dep. Health, New York City, N.Y.). *Bull. NY Acad. Med.* 51(2): 263-271, 1975.

During the 14-yr period from 1960 through 1973, a total of 3,643 cases of schistosomiasis were reported in New York City. Between 1960 and 1973 the number of cases reported annually declined by 66.4%. This decline is closely tied to changing immigration patterns and to the development of programs for the control and treatment of schistosomiasis in Puerto Rico. A rise in reported cases was noted in Brooklyn in the last 3 years, due to infections among the growing number of recent Yemenite immigrants occupying the Bushwick area. The majority of schistosomiasis cases have been reported from Brooklyn, Manhattan and the Bronx, the areas which harbor most recent immigrants from endemic areas. The majority of cases were diagnosed in adults between 20 and 44 yr of age (2,027 cases, 76.5%). Children less than 15 yr of age accounted for only 10.8%. This age distribution reflects the age distribution of the immigrant population. For the 12-yr period from 1960 through 1971, the male-to-female ratio was 1.9:1. Among Yemenites in Brooklyn, this ratio was 12.6:1. An analysis of surnames between 1970 and 1973 revealed that patients with Spanish surnames have comprised about 90% of all cases in the Bronx. In Manhattan the proportion with Spanish surnames declined from 92.3% in 1970 to 77.2% in 1973. The vast majority of cases of schistosomiasis reported in New York City during the period of study were *Schistosoma mansoni* infections. Several cases of *Schistosoma haematobium* were reported, but none of *Schistosoma japonicum*. It is concluded that the epidemiologic pattern of schistosomiasis in New York City will be determined by future immigration and by the effectiveness of schistosomiasis control programs in endemic areas.

1584 TINIDAZOLE IN THE TREATMENT OF INTESTINAL AMOEBIASIS. *(E.)* Islam, N.; Hasan, M. (Inst. Postgrad. Med. Res., Dacca, Bangladesh). *Curr. Ther. Res.* 17(2):161-165, 1975.

Fifty patients with chronic intestinal amebiasis (35 males and 15 females, aged 17-60 yr) were treated with tinidazole. The duration of symptoms varied from less than 2 months to 2 yr. Nine cases were asymptomatic and 4 had associated diseases so that the duration of symptoms could not be ascertained. Tinidazole was given as a single dose of 2000 mg/day after meals for 3 consecutive days. Parasitic cure was obtained in 49 cases, clinical cure in 44, both clinical and parasitic cure in 43 (86%). Side effects included anorexia (5 cases), nausea (2), general malaise (10), and metallic taste in the mouth (3). No withdrawal of therapy was necessitated by these minor side effects. It is suggested that the advantages of a daily single dose of tinidazole as opposed to 2 or 3 divided doses are obvious, provided side effects are minimal. The single dose has the theoretical advantage of having a higher serum peak level with perhaps greater amebicidal effect. These results indicate that tinidazole used as described may not be inferior to metronidazole. In 4 of these cases, hepatic amebiasis was present, but did not necessitate lengthening the therapy except in one case, where the therapy was repeated. It is suggested that this regime may be sufficient for the treatment of mild to moderate hepatic amebiasis.

In moderately severe cases treatment may have to be prolonged along with aspiration until satisfactory control is obtained.

1585 HEPATIC INVOLVEMENT IN AMOEBIASIS. *(E.)* Kasliwal, R. M. (Med. Coll., Jaipur, India). *Am. J. Proctol.* 26(1):43-48, 1975.

The question of whether some cases of so-called chronic hepatitis and cirrhosis have an amebic background was investigated using a group of 60 patients. These patients were divided into 4 groups. Group I showed hepatomegaly ranging from 1 to 3 fingers enlarged (19 patients); Group II, amebic hepatitis (13); Group III, liver abscess, amebic (5); and group IV, cirrhosis (23). An Indirect Hemagglutination Test (IHA) was performed on each patient. Of the 13 patients with chronic hepatitis suspected to be of amebic origin, 10 showed a positive IHA test. Of the 23 patients with cirrhosis, 8 showed a positive IHA test. In the hepatomegaly cases of non-amebic origin, all 19 cases had a negative IHA test and all 5 cases of liver abscess of amebic origin had a strongly positive IHA test. Of the 8 cirrhotics with positive IHA test results, 4 improved very much and were discharged as cured on anti-infective, anti-amebic and steroid therapy. Two others showed partial improvement and 2 responded initially but, after 2 1/2 months, did not show sustained improvement. It is noted that in those cases where the IHA test was positive in high dilutions, the patients did not show much improvement, whereas in those in whom the IHA test was positive in low titers, the prognosis was better and the patients responded to treatment. Thus the IHA test may be of prognostic as well as diagnostic value. These findings suggest the possibility that some of the cases of chronic hepatitis and cirrhosis found particularly in an endemic area may have an amebic background. It is urged that in patients with chronic hepatitis and cirrhosis where an obvious etiological background is not found, there be a thorough exploration for a possible amebic infection.

1586 RECENT EXPERIENCE WITH HEPATIC AMEBIASIS. SUCCESSFUL TREATMENT WITH METRONIDAZOLE. *(E.)* Wadlington, W. B.; Faber, R.; O'Neill, Jr., J. A. (Vanderbilt Univ. Med. Cent., Nashville, Tenn.). *Clin. Pediatr.* 14(2):163-170, 1975.

A 4-yr old girl was admitted with a 3 week history of weight loss and fever and a one-week history of jaundice. The liver was palpable 5 cm below the costal margin. No ova or parasites were seen in the stool. A chest x-ray showed a slightly elevated right diaphragm with infiltrates in the right lower lobe. Abdominal films demonstrated an enlarged liver. Liver scan showed a right posterosuperior filling defect, interpreted as an abscess. The abscess was aspirated and when stained the pus showed *Entamoeba histolytica*. The child received metronidazole (50 mg/kg/day, or 750 mg/day, for 5 weeks). She became afebrile in 4 days and began to gain weight. A liver scan performed 5 weeks after initial studies was normal. A 5 yr old boy was hospitalized in shock, jaundice, and extreme lethargy. Three weeks earlier he had complained of right upper quadrant pain. The liver was tender, large and extended almost to the right iliac crest. Abdominal films demonstrated an enlarged liver. Liver scan demonstrated a large filling defect of the right lobe of the liver which was aspirated and the stained material showed both *E. histolytica* and gram-positive cocci. The culture grew out *Staphylococcus aureus*. He received metronidazole (50 mg/kg/day, or 750 mg/day) and methcillin (100 mg/kg/day). Therapy was continued for 1 month. After the tenth treatment day the fever left and the child rapidly improved. However, 3 months passed before the liver scan was normal. Shorter courses of therapy than used in these 2 patients are usually recommended. The schedule for the patients was based on their clinical course and the time the liver scan took to improve. Neither patient displayed any toxic effects.

1587 EFFECT OF HYCANTHONE ON VARIOUS DEVELOPMENTAL STAGES OF Schistosoma mansoni IN MICE. *(Fr.)* Lapierre, J.; Holler, C.; Lebas-Saison, E.; Le Prious, B.; Roose, A. (Cochin Hosp., Paris, France). *C.R. Soc. Biol. (Paris)* 168(4/5):452-455, 1974.

1588 EFFECT OF (5-NITRO-2-THIAZOLYL)-1-IMIDAZOLIDINE THIONE-2 (R.P. 26354) ON EXPERIMENTAL SCHISTOSOMIASIS IN LABORATORY ANIMALS. *(Fr.)* Benazet, F.; Leroy, J. P. (Res. Lab., Rhone-Poulenc Chem. Co., Vitry-sur-Seine, France). *Bull. Soc. Pathol. Exot.* 67(3):287-297, 1974.

1589 EPIDEMIOLOGICAL STUDY OF INTESTINAL AND URINARY SCHISTOSOMIASIS IN THE LUFIRA DAM REGION. 5. EFFECT OF HELMINTHIASIS ON THE HEALTH OF SUBJECTS EXCRETING Schistosoma mansoni EGGS. *(Fr.)* Raccurt, C.; Rippert, C.; Grimaud, J. A. (Fac. Med., Natl. Univ. Zaire, Kinshasa). *Bull. Soc. Pathol. Exot.* 67(4):402-424, 1974.

1590 A SINGLE INTRAMUSCULAR DOSE OF HYCANTHONE: THERAPEUTIC RESULTS OBTAINED ON SUBJECTS EXCRETING S. mansoni EGGS TREATED AT KAPOLOWE (LUFIRA DAM REGION, REPUBLIC OF ZAIRE). *(Fr.)* Raccurt, C.; Grimaud, J. A.; Ripert, C. (Fac. Med., Natl. Univ. Zaire, Kinshasa). *Bull. Soc. Pathol. Exot.* 67(4):425-435, 1974.

1591 OWN EXPERIENCES WITH TREATMENT OF HEPATIC ECHINOCOCCUS. *(Pol.)* Gerlach, Z.; Gerlach, A. (Al-Attaf Hosp., Algeria). *Wiad. Lek.* 27 (14):1259-1263, 1974.

1592 NEW DATA ON THE INTERACTIONS BETWEEN THE BILE DUCT DWELLERS, Fasciola hepatica(TREMATODA) AND Hymenolepis microstoma (CESTODA), IN MICE. *(E.)* Gleason, L. N. (Sch. Public Health, Chapel Hill, N.C.). *J. Elisha Mitchell Sci. Soc.* 90(2):58-63, 1974.

1593 GIARDIASIS, LOCAL IMMUNE DEFENSE AND RESPONSES. *(E.)* Popovic, O.; Pendic, B.; Paljm, A.; Andrefevic, M.; Trpkovic, D. (Clin. Hosp., Belgrade, Yugoslavia). *Eur. J. Clin. Invest.* 4(5): 380, 1974.

1594 TREATMENT OF CHILDREN WITH GIARDIASIS USING A LIQUID PREPARATION OF METHYLMERCADONE (NIFURATEL), ADMINISTERED ORALLY, FOR FIVE DAYS. *(Por.)* de Silva, M. A. P. (Santa Casa Fac. Med. Sci., Sao Paulo, Brazil). *Rev. Bras. Clin. Ter.* 3(12):449-452, 1974.

1595 ETOFAMIDE AS A NEW WAY FOR TREATING CHRONIC INTESTINAL AMEBIASIS. *(Por.)* Alterio, D. L. (Sao Paulo Med. Sch., Brazil). *Rev. Bras. Clin. Ter.* 3(12):453-456, 1974.

1596 AMEBIC LIVER ABSCESS IN CHILDHOOD. AN ATYPICAL CASE IN AN 18-MONTH-OLD CHILD. *(E.)* Jessee, W. F.; Ryan, J. M.; Fitzgerald, J. F.; Grosfeld, J. L. (Indiana Univ. Med. Cent., Indianapolis). *Clin. Pediatr.* 14(2):134-135, 1975.

1597 INCIDENCE AND IMPORTANCE OF COMMON INTESTINAL PARASITES AMONG THE POPULATION OF THE PUNJAB IN NORTH-WEST-INDIA. *(E.)* Stickl, H.; Werner, G. T.; Sareen, D. K. (Bavarian State Inst. Vaccinations, Munich, Germany). *Infection* 2(3): 118-122, 1974.

See also, 1149, 1194, 1197.

GENERAL

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See also, 1105, 1136, 1150, 1225, 1271.

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PRE-CLINICAL SCIENCES

MORPHOLOGY, CELLULAR STRUCTURE AND HISTOCHEMISTRY

1598 THE EFFECT OF STARVATION AND REFEEDING ON CELL POPULATION KINETICS IN THE RAT SMALL BOWEL MUCOSA. *(E.)* Aldewach, H. S.; Wright, N. A.; Appleton, D. R.; Watson, A. J. (Dep. Pathol., Univ. Newcastle upon Tyne, England). *J. Anat.* 119(1):105-121, 1975.

The effects of starvation and refeeding on the proliferative indices, growth fraction, and cell cycle time in the rat small bowel were studied. Three-month-old male Wistar rats were starved continuously for 96 hours then allowed free access to food for 0-48 hr. Some animals were injected with vincristine before refeeding and all were injected with tritiated thymidine (0.5 μCi/g) 1 hr before death. Morphometric parameters, proliferative and/or labeling indices, and cell cycle parameters were measured in axially sectioned crypts from each animal. The crypt cell population showed a small reduction during starvation. The growth fraction remained constant but the total number of proliferating cells/crypt fell as a consequence of the reduction in crypt population. Both labeling and mitotic indices fell throughout the starvation period. Cell cycle time, measured by a stathmokinetic technique using vincristine, increased from 10.4 hr in control rats to 14.7 hr after starvation. Upon refeeding, the proliferative indices rose, while, after a small initial fluctuation, the growth fraction remained constant. The crypt population remained substantially unchanged and 16 hr after refeeding, the cell cycle time was reduced to 6.5 hr. The data indicate that the hypoproliferative response to starvation is mediated solely by an increase in cell cycle time, and the response to refeeding is interpreted in terms of a reduction in the cell cycle time. Changes in the size of the proliferating population do not appear to play an important role in either response, although it is not possible to entirely exclude the presence of resting cells in the proliferative compartment itself.

1599 GRANULE RELEASE FROM ENDOCRINE CELLS IN ACIDIFIED HUMAN DUODENAL BULB: AN ELECTRON MICROSCOPE STUDY OF BIOPSY MATERIALS. *(E.)* Osaka, M.; Sasagawa, T.; Fujita, T. (Niigata Univ. Sch. Med., Japan). *Arch. Histol. Jpn.* 37(1):73-94, 1974.

A study was made of the types, fine structure, and distribution of endocrine elements in the human duodenal bulb and the responses of the cells to dilute HCl in order to identify those cells producing anti-acidic hormones. Biopsies (by duodenoscopy) of the mucosal tissue from the duodenal bulb of 6 patients were prepared for histological study. Four other patients had 40 ml of 0.1 N HCl infused into the duodenal bulb, then biopsies were taken after 5 min. Immunofluorescence techniques were used to identify hormones with gastroenteric endocrine cells. A greater number of endocrine cells were found in the duodenal bulb than in the lower part of the duodenum; 9 types were identified by their fine structures. Following acidification (pH 1.5) of the duodenal bulb, granule release by emiocytosis was observed under the electron microscope in 4 of the types. The granules appeared in pairs enclosed in a dumbbell-shaped invagination of plasma membrane of the gastroenteric endocrine cell. The granule release was considered a sign of accelerated release of secretory granules. The hormones produced by D_1, D_{1l}, L and M cell types were proposed to be anti-acidic agents such as secretin, enterogastrone, cholecystokinin-pancreozymin and enteroglucagon, and the are assumed to be of great significance in the homeostatic control of the secretory activities of the gut (including liver and pancreas) caused by the ingestion of food.

1600 MEMBRANE UNBOUND CRYSTALS IN PANCREATIC ACINAR CELLS OF THE MOUSE. *(E.)* Machino, M.; Hoshino, K. (Fac. Med., Univ. Manitoba, Winnipeg, Canada). *Virchows Arch.* [*Zellpathol.*] 17(3):261-267, 1975.

The intracytoplasmic crystals found in the pancreatic acinar cells of the mouse were studied by electron microscopy. Pancreatic sections were taken from 4-month-old male C57BL mice. The basal cytoplasmic areas of the acinar cells frequently contained elongated, conical, oblong, rectangular, or rhomboid shaped crystals in the interspaces of the rough endoplasmic reticulum. The crystals were less than 5 μm in length and more than 0.1 μm in thickness and exhibited low to moderated electron densities. These crystals were detected in at least 1 cell in each ultrathin section. A parallel pattern formed by dense lines and less dense interspaces was observed in different crystal matrices. The center-to-center distances between the dense lines measured 100-150 Å. The crystal surfaces were devoid of any limiting membrane so that the outer aspects of the attached ribosomes of the endoplasmic reticulum were in direct contact with the crystal. On occasions when the ribosomes of the endoplasmic reticulum or free ribosomes were in close proximity to, but not in contact with the crystals, the ribosomes were connected with the surface of the crystals by filaments of 50-100 Å in thickness. The terminal portions of these filaments merged into the homogeneous crystal matrix, the heterogeneous portions of the matrix being occupied by tangled filaments or ribosomes. There was no indication that the crystal materials originated from a membrane element of the rough endoplasmic reticulum, Golgi sacs, lysosomes, mitochondria, nuclei, or degenerative organelles. The crystals of the acinar cells may be composed partially or wholly of protein, which seems to have a very slow turnover rate.

1601 RECOVERY OF PANCREAS FROM MILD PUROMYCIN-INDUCED INJURY. A HISTOLOGIC AND ULTRASTRUCTURE STUDY IN RATS. *(E.)* Longnecker, D. S.; Crawford, B. G.; Nadler, D. J. (Dartmouth Med. Sch., Hanover, N.H.). *Arch. Pathol.* 99(1):5-10, 1975.

Light and electron microscopic studies were conducted to characterize pancreatic acinar cell recovery follow-

ing treatment of male Wistar rats with puromycin (40 mg/kg/hr i.p. for 4 hr). Peak pancreatic cell damage occurred after 12-24 hr at which time electron microscopy showed numerous autophagic vacuoles and intracisternal granules in the cells. By 48 hr, these were largely cleared from surviving cells although the intracisternal granules persisted in isolated acinar cells as long as 144 hr. At 24 hr, there was debris in the acinar lumina and interstitial space. It is concluded that some acinar cells injured by puromycin may survive and be restored to normal structure; that surviving acinar cells can extrude autophagic vacuoles; and that necrotic acinar cells are replaced by regeneration following puromycin-induced injury in rats.

8602 FENESTRATED ENDOTHELIUM OF THE LIVER SINUSOIDS OF THE GUINEA PIG AS REVEALED BY SCANNING ELECTRON MICROSCOPY. *(E.)* Itoshima, T.; Kobayashi, T.; Shimada, Y.; Murakami, T. (Okayama Univ. Med. Sch., Japan). *Arch. Histol. Jpn.* 37(1): 15-24, 1974.

The endothelium of the liver sinusoids of the guinea pig was studied by scanning electron microscopy. Specimens were fixed with 2% glutaraldehyde by a perfusion technique and were treated with a revised tannin-osmium method which avoids coating of the specimen with metal. The endothelium contained numerous intracellular and some intercellular fenestrae, the size and distribution of which varied with the position within the lobule. Larger fenestrae (0.5-3.0 μ in diameter) occurred in the endothelium near the central veins. The fenestrae became smaller and fewer in number toward the periphery of the lobules, measuring less than 0.3 μ in diameter close to the distributing veins. In addition, groups of micropores formed sieves in the concavities among the reticular crests of the endothelial sheet. The nuclear swelling as well as the cytoplasmic crests possessed small pits which most likely represented pinocytotic invagination. In the periportal area, the sinusoidal wall was partially composed of cells, presumably Kupffer cells, which had processes extending into the lumen. Cells with long processes extending over microvilli of hepatic cells, which were thought to be the fat-storing cells of Ito, could be seen through the large fenestrae just below the endothelium. The larger fenestrae situated near the central veins may function to supply a relatively greater quantity of blood, and hence nutrients, to satisfy the metabolic demands of this area.

8603 HEPATIC FINE STRUCTURE IN YOUNG AND AGING RATS TREATED WITH OXANDROLONE: A MORPHOMETRIC STUDY. *(E.)* Schmucker, D. L.; Jones, A. L.; (San Francisco VA Hosp., Calif.). *J. Lipid Res.* 16(2):143-150, 1975.

Morphometric techniques were used to study the fine structural changes induced by oxandrolone in the livers of normolipidemic young rats and hyperlipidemic aging rats (retired breeders). Young adult and retired breeder male Sprague-Dawley rats were given oxandrolone (100 mg/kg/day) in corn-oil margarine for 21 days p.o., while control animals received the vehicle alone. The animals were then fasted for 24 hr, and given another dose of the drug or vehicle 12 hr before killing. In the young rats, oxandrolone reduced (nonsignificantly) the unit volume of the extrahepatocyte space and increased the average hepatocyte unit volume by a similar amount. The mean cytoplasmic volume fraction was greater in the experimental than in the control livers; this was attributed to a greater volume fraction of cytoplasm in the parenchymal cells of the former. There were no significant changes in the volume densities of the mitochondria, microbodies, lysosomes, or dense bodies. After oxandrolone administration, the mean surface density of the smooth endoplasmic reticulum, rough endoplasmic reticulum, and Golgi apparatus increased by 35%, 25% and 19%, resp. In the retired breeders, oxandrolone did not significantly affect the volume densities of the extrahepatocyte space or hepatocytes, and an observed reduction in the mean volume fraction of the biliary space was not significant. The experimental liver cells appeared to contain less cytoplasm than the control cells. There was a significant change in the volume fraction of microbodies in the livers of oxandrolone-treated animals. Although the hepatocytes in the control retired breeders contained significantly less smooth endoplasmic reticulum than the young controls, oxandrolone increased the surface density of the smooth endoplasmic reticulum in the older animals to a level 68% above the control value; thus there was no significant difference in the surface densities between the young and aged animals. Oxandrolone also increased the surface density of the rough endoplasmic reticulum and Golgi apparatus by 21% and 41%, resp. The data suggest that the age-related fine structural changes are not the result of irreversible alterations in the genome or translation-transcription apparatus; they may represent secondary reactions to extrahepatic and/or endocrine metabolic changes.

8604 ULTRASTRUCTURE AND FUNCTION OF THE PAROTID GLAND IN NORMAL RATS. *(Rus.)* Tsoi, G. K.; Nechaeva, N. V.; Eletskii, Iu. K. (N. I. Pirogov 2nd Moscow Med. Inst., USSR). *Arkh. Anat. Gistol. Embriol* 67(7):69-74, 1974.

8605 DEVELOPMENT OF ENTEROCHROMAFFIN AND DOPAMINE CELLS IN THE GASTROINTESTINAL TRACT OF THE CALF. *(E.)* Ahonen, A.; Penttila, A. (Dep. Anat., Univ. Helsinki, Finland). *Experientia* 30(12):1460-1462, 1974.

8606 OBSERVATIONS ON THE SUBMUCOUS PLEXUS AND MUCOSAL ARTERIES OF THE DOG'S STOMACH AND FIRST PART OF THE DUODENUM. *(E.)* Piasecki, C. (R. Free Hosp. Sch. Med., London, England). *J. Anat.* 119(1):133-148, 1975.

8607 EFFECT OF PYRIDINE DISSOLVED IN WATER ON THE GASTRIC AND INTESTINAL MUCOSA OF EXPERIMENTAL ANIMALS. *(Rus.)* Kondratiuk, V. A. (Ternopol Med. Inst., USSR). *Gig. Sanit.* (11):87-88, 1974.

1608 THE DISTRIBUTION OF WHITE BLOOD CELLS IN THE GASTRIC AND HEPATIC CAPILLARIES IN THE COURSE OF HYDROCORTISONE ADMINISTRATION. ELECTRON MICROSCOPIC STUDY. *(E.)* Dragomir, C. T.; Tomescu, E.; Cotutiu, C.; Ungureanu, D. (Victor Babes Inst., Bucharest, Rumania). *Arch. Roum. Pathol. Exp. Microbiol.* 33(2):191-194, 1974.

1609 ULTRASTRUCTURE OF ENDOCRINE CELLS IN THE HUMAN GASTRIC MUCOSA. *(Ger.)* Mitschke, H. (Pathol. Inst., Univ. Hamburg, Germany). *Verh. Dtsch. Ges. Pathol.* 57:446, 1973.

1610 THE EFFECTS OF PARA-CHLOROMECURIBENZENE SULFONATE UPON ULTRASTRUCTURE OF CANINE GASTRIC SURFACE CELLS. *(E.)* Zalewsky, C. A.; Moody, F. G.; Simons, M. A. (Univ. Utah Med. Cent., Salt Lake City). *Fed. Proc.* 34(3):453, 1975.

1611 HEXAGONAL CRYSTALLINE BODIES OF IgA IN THE CYTOPLASM OF THE HEPATOCYTES. *(E.)* Bednar, B.; Bliazek, J.; Brodanova, M.; Hermansky, F.; Smetana, K. (Fac. Med., Charles Univ., Prague, Czechoslovakia). *Pathol. Eur.* 9(3):193-198, 1974.

1612 LIVER ULTRASTRUCTURE AND LIPID COMPOSITION OF LIVERS FROM OBESE HENS WITH HEMORRHAGIC LIVERS. *(E.)* Wills, J. R.; Savage, J. E.; Tumbleson, M. E. (Sch. Vet. Med., Univ. Missouri, Columbia). *Fed. Proc.* 34(3):933, 1975.

1613 MINOR COMPONENTS OF RAT LIVER RIBOSOMAL RNA AS POSSIBLE INTERMEDIATES IN THE DEGRADATION OF 28S RNA IN VIVO. EFFECTS OF PARTIAL HEPATECTOMY AND STARVATION ON THEIR RELATIVE AMOUNTS. *(E.)* Inoue, T.; Takagi, M.; Umemura, Y.; Yamaguchi, H. (Lab. Radiat. Genet., Fac. Agric., Univ. Tokyo, Japan). *Agric. Biol. Chem.* 38(12):2457-2464, 1974.

1614 THE LOCATION OF BILE ACIDS IN THE HEPATOCYTE. I. *(Ger.)* Leuschner, U.; Alfurayh, A.; Uhlmann, W.; Wildgrube, H. J.; Erb, W. (Cent. Intern. Med., Johann Wolfgang Goethe Univ., Frankfurt/Main, Germany). *Z. Gastroenterol.* 12(2):95-102, 1974.

1615 REGENERATION AND FUNCTIONAL RECOVERY OF CANINE INTESTINAL MUCOSA FOLLOWING INJURY CAUSED BY FORMALIN. *(E.)* Stamm, B.; Mirkovitch, V.; Winistorfer, B.; Robinson, J. W. L.; Ozzello, L. (Canton. Univ. Hosp., Lausanne, Switzerland). *Virchows Arch.* [*Zellpathol.*] 17(2):137-148, 1974.

See also, 1811, 1812, 1870, 2300.

ABSORPTION—EXCRETION—TRANSPORT

1616 TRANSPORT OF H^+ AND OTHER ELECTROLYTES ACROSS ISOLATED GASTRIC MUCOSA OF THE RABBIT. *(E.)* Fromm, D.; Schwartz, J. H.; Quijano, R. (Harvard Med. Sch., Boston, Mass.). *Am. J. Physiol.* 228(1):166-171, 1975.

Ion transport and the relation between luminal acidification and organic acid production, metabolic CO_2 production and transmural pH gradients was studied in isolated gastric fundic mucosa of New Zealand white male rabbits. Both the luminal and serosal surfaces were bathed in a Ringer solution free of bicarbonate and CO_2. Luminal acidification resulted primarily (at least 90%) from H^+ secretion rather than from organic acid accumulation (based on measurements of pyruvate and lactate), or from the accumulation of CO_2. The availability of O_2 and endogenous substrate was a limiting factor for spontaneous acid secretion, while the endogenous production of CO_2 was not a limiting factor. The differential appearance of CO_2 on the luminal and serosal borders of the mucosa suggested that a fraction of metabolic CO_2 is hydroxylated in the cell. CO_2 appeared on the serosal side at a greater rate than on the luminal side, and the difference approximated the rate of luminal acidification. This is most likely due to the cellular formation of bicarbonate that preferentially diffuses across the serosal border. Lowering the luminal pH to 4.5 caused a significant decrease in luminal acidification, and at pH 3.0 acidification ceased. In addition to net H^+ secretion, there was also net Cl^- and net Na^+ secretion, while net K^+ secretion was negligible. It was suggested that since luminal changes in H^+ concentration affected the rate of acid secretion and serosal changes did not, the gradient produced by H^+ secretion probably occurs at the luminal border.

1617 MECHANISMS OF ACID DISPOSAL IN CANINE DUODENUM. *(E.)* Dorricott, N. J.; Fiddian-Green, R. G.; Silen, W. (Harvard Med. Sch., Boston, Mass.). *Am. J. Physiol.* 228(1):269-275, 1975.

The ability of the canine duodenum distal to the major pancreatic duct, and therefore free from pancreatic, biliary, and Brunner's gland secretions, to dispose of acid was studied. Particularly examined were the capacity and mechanisms of disposal and whether they are load or concentration dependent. Distal duodenal pouches chronically exteriorized from the intestinal stream (those studied between 1 week and 12 months postoperatively) were perfused in 7 conscious mongrel dogs by a constant-circulation technique using isosmolar solutions containing 60-120 mM

HCl. The rate of disappearance of hydrogen ions (H^+) and net ion movements were measured. In both experimental groups (3 hour experiments and 30 min experiments) there was a loss of H^+ from the perfusate accompanied by a gain of sodium, potassium, and chloride, together with an increase in volume and a decrease in osmolality. The acid loss from any single area of the duodenum is a function of the H^+ to which it is exposed. The H^+ lost by neutralization was separated from that lost by diffusion by calculations based on the assumption that the observed changes in osmolality were the result of neutralization by bicarbonate: 67% of the acid was lost by neutralization, the rest back-diffusing in a 1:1 exchange for diffusable sodium. The ability to dispose of acid was impaired in those pouches which were chronically exteriorized and repeatedly exposed to acid, due to decreased secretion of bicarbonate.

1618 DEMONSTRATION OF THE RAPID ACTION OF PURE CRYSTALLINE 1α-HYDROXY VITAMIN D_3 AND 1α,25-DIHYDROXY VITAMIN D_3 ON INTESTINAL CALCIUM UPTAKE. *(E.)* Toffolon, E. P.; Pechet, M. M.; Isselbacher, K. (Harvard Med. Sch., Boston, Mass.). *Proc. Nat. Acad. Sci. USA* 72(1):229-230, 1975.

The effects of crystalline 1α-hydroxy vitamin D_3 (1α-OH-D_3) and 1α,25-dihydroxy vitamin D_3 (1α,25-$(OH)_2$-D_3) on intestinal uptake of ^{45}Ca were studied in Sprague-Dawley rats (mothers and male pups). Everted duodenal gut sacs from rachitic rats were studied after the rats had received 0.1 μg of either 1α-OH-D_3 or 1α,25-$(OH)_2$-D_3 *via* the jugular vein. Peak ^{45}Ca uptake was observed 1 hr after administration and both crystalline analogs were of comparable potency. The rapid induction of intestinal calcium transport by the crystalline vitamin D_3 analogs suggests a direct effect on the intestinal mucosal membrane, but this has not been demonstrated *in vitro*.

1619 GALACTOSE TRANSPORT ACROSS THE SEROSAL BORDER OF RABBIT ILEUM AND ITS ROLE IN INTRACELLULAR ACCUMULATION. *(E.)* Holman, G. D.; Naftalin, R. J (Dep. Physiol., Univ. Leicester, England). *Biochim. Biophys. Acta* 382(3):230-245, 1975.

To determine the role of the serosal border of the ileum in the transport and accumulation of sugar in the cell, unidirectional fluxes of D-galactose across the brush and serosal border of the rabbit ileum were determined using the method of Naftalin and Curran. With Ringer's solution (Na) = 75 mEq, the K_m for galactose influx across the brush-border was 5 mM; with 0.1 mM ouabain present, the K_m was 50 mM, but the V (2.0 μM/cm^2/hr) remained unaltered. The Michaelis parameters for galactose influx across the serosal border were K_m = 59 and V = 4.7 μM/cm^2/hr, and for efflux, K_m = 85 mM and V = 6.8 μM/cm^2/hr. 2-Deoxy-D-glucose and methyl β-D-glucopyranoside inhibited galactose entry exclusively at the serosal and mucosal borders, resp., while 3-O-methyl-D-glucose inhibited galactose influx at both borders. Ouabain (0.1 mM) increased the K_1 of 3-O-methylglucose for the mucosal galactose transport system from 50-150 mM, while the K_1 of 3-O-methylglucose for the serosal transport system (100 mM) was unaltered. The inhibition of mucosal galactose transport by ouabain or by competition with other sugars resulted in reciprocal increase in the exit permeability and a decrease in the entry permeability. The inhibition of serosal galactose transport resulted in inhibition of both the entry and exit permeability, particularly the former. There was a small degree of permeability asymmetry at the serosal border to galactose which was reduced by ouabain or removal of Na^+ from the Ringer. The uptake of ^{14}C-labeled galactose from the serosal solution into the tissue was also inhibited by the addition of ouabain or removal of Na^+. Thus, there appears to be a weak active transport system for galactose at the serosal border. The net transepithelial flux was sufficiently high and the serosal permeability to galactose sufficiently low to be consistent with the view that galactose is concentrated within the tissue fluid after convection across the mucosal border because it is reflected at the serosal boundary.

1620 ABSORPTION OF VITAMIN B_{12} IN RATS WITH JEJUNO-ILEAL TRANSPOSITION. *(E.)* Gronqvist, B. (Univ. Hosp., Uppsala, Sweden). *Acta. Chir. Scand.* 140(8):637-642, 1974.

The propulsive motility of the small intestine and the uptake of vitamin B_{12} by the small intestinal wall were studied in rats 2 weeks after jejuno-ileal transposition. Sprague-Dawley rats were sham-operated (control) or subjected to jejuno-ileal transposition, in which the jejunum and ileum were interchanged so that the ileum was placed proximally and the jejunum distally in the continuity of the intestine. The propulsive motility of the gastrointestinal tract was measured in both groups before and after surgery by recording the distribution of catheter-introduced sodium chromate in the gastrointestinal tract. The distribution of vitamin B_{12} in the gastrointestinal tract was measured in the same way using vitamin B_{12} labeled with ^{57}Co. Postoperative weight gain in the transposed animals was somewhat less than in the sham-operated rats, while the positions of the anastomoses showed little variation between the two groups. The amount of vitamin B_{12} remaining in the stomach at the end of the experimental period (2 weeks postoperatively) showed no significant difference between the 2 groups, but the controls showed a significantly greater uptake of the vitamin in the small-intestinal wall and a significantly greater content of vitamin B_{12} in the solution used to flush the small intestine at the end of the experiment. The distribution of radioactive chromate and vitamin B_{12} were essentially similar, although vitamin B_{12} had a somewhat more proximal distribution than the corresponding chromate. The data indicate that in the rat a transposed ileal segment improves its capacity to take up vitamin B_{12} with time after the operation, i.e., that a functional normalization takes place.

1621 EFFECTS OF SODIUM TAUROCHENODEOXYCHOLATE AND SODIUM TAUROCHOLATE ON CHOLESTEROL ABSORPTION IN THE RAT. *(E.)* Raicht, R. F.; Cohen, B. I.; Mosbach, E. H. (Manhattan VA Hosp., New York, N.Y.). *Gastroenterology* 67(6):1155-1161, 1974.

The effect of dietary sodium taurochenodeoxycholate and sodium taurocholate on cholesterol absorption was studied in male Sprague-Dawley rats which were fed a commercial diet to which was added 5% corn oil and small amounts of cholesterol. This diet contained 1.61 mg/g of cholesterol and 0.62 mg/g of β-sitosterol. The diet was supplemented with sodium taurocholate (0.5%) or sodium taurochenodeoxycholate (0.5%). The rats in each group had similar food intakes (27 g/day), similar fecal outputs (8 g/day), and gained similar amounts of weight (100 g). Taurochenodeoxycholate did not significantly alter plasma cholesterol concentration, but it did increase hepatic cholesterol content (control rats, 2.5 mg/g; taurochenodeoxycholate-fed rats, 4.5 mg/g). Taurocholate feeding significantly increased plasma cholesterol concentration (control rats, 86 mg/100 ml; taurocholate-fed rats, 114 mg/100 ml), as well as increasing liver cholesterol concentration (8.0 mg/g). The differential effect of taurocholate and taurochenodeoxycholate on cholesterol absorption has several implications. The fact that taurochenodeoxycholate does not increase cholesterol absorption suggests that this bile acid directly affects 3-hydroxy-3-methylglutaryl CoA reductase, the rate-limiting enzyme of cholesterol synthesis. It also suggests that hepatic cholesterol accumulation in taurochenodeoxycholate-fed rats may be due to a direct effect of the bile acid on hepatic cholesterol metabolism. The greater accumulation with taurocholate, however, is due, at least in part, to the increased absorption seen with this bile acid.

1622 SURFACE AREA, OXIDATION REDUCTION POTENTIALS, AND pH IN THE GASTROINTESTINAL TRACT OF CONVENTIONAL AND GNOTOBIOTIC BEAGLES. *(E.)* Stevens, N. C.; Heneghan, J. B. (Louisiana State Univ. Sch. Med., New Orleans). *Fed. Proc.* 34(3): 475, 1975.

1623 STUDIES ON THE METABOLIC FATE OF ^{32}P-LABELLED EMULSIFIER YN IN THE MOUSE, GUINEA-PIG AND FERRET. *(E.)* Phillips, J. C.; Gaunt, I. F.; Gangolli, S. D. (Br. Ind. Biol. Res. Assoc., Carshalton, England). *Food Cosmet. Toxicol.* 13(1): 23-30, 1975.

1624 INTERPRETATION OF TRANSIENT VOLTAGE RESPONSE OF EPITHELIAL TISSUES TO STEP CURRENTS. *(E.)* Sanders, S. S.; Rehm, W. S. (Univ. Alabama Med. Cent., Birmingham). *Fed. Proc.* 34(3): 286, 1975.

1625 EFFECTS OF BILE SALT ON ION TRANSPORT IN THE CANINE GASTRIC MUCOSA. *(E.)* Kuo, Y. J.; Shanbour, L. L. (Univ. Texas Med. Sch., Houston). *Fed. Proc.* 34(3):286, 1975.

1626 THE EFFECT OF AGE ON THE GASTROINTESTINAL ABSORPTION OF AMINO ACIDS AND PEPTIDES. *(E.)* Ludorf, J.; Gallo-Torres, H. E. (Roche Res. Cent., Nutley, N.J.). *Fed. Proc.* 34(3):453, 1975.

1627 EFFECT OF PHENOXAZINE COMPOUNDS ON AMINO ACIDS ACCUMULATION IN RAT SMALL INTESTINE. *(E.)* Nakamura, Y.; Shibata, K.; Asai, W.; Minoura, H.; Nagata, Y. (Lab. Biochem., Fac. Agric., Gifu Univ., Naka Kagamigahara, Japan). *Agric. Biol. Chem.* 39(1):113-117, 1975.

1628 INTESTINAL ABSORPTION OF TETRAHYDROBIOPTERIN AND PTEROIC ACID IN MAN: A COMPARISON OF MODELS FOR THE TRANSPORT OF FOLATES ACROSS THE SMALL INTESTINE. *(E.)* Blair, J. A.; Ratanasthien, K.; Leeming, R. J. (Dep. Chem., Univ. Aston, Birmingham, England). *Gut* 15(10):832, 1974.

1629 D-AMINO ACID-INDUCED TRANSMURAL POTENTIAL CHANGES IN RAT SMALL INTESTINE. *(E.)* Nakamura, Y.; Yasumoto, K.; Sugiyama, K.; Mitsuda, H. (Fac. Agric., Kyoto Univ., Japan). *Agric. Biol. Chem.* 38(11):2149-2155, 1974.

1630 INFLUENCE OF DIETARY AMINO ACID COMPOSITION ON RATES OF INTESTINAL ABSORPTION. *(E.)* Herbert, J. D.; Coulson, R. A. (Louisiana State Univ. Med. Cent., New Orleans). *Fed. Proc.* 34(3):916, 1975.

1631 THE EFFECT OF SUCROSE FEEDING ON INTESTINAL TRANSPORT OF VARIOUS SUGARS IN TWO RAT STRAINS. *(E.)* Hallfrisch, J.; Reiser, S.; Michaelis, IV, O. E.; Putney, J. (U.S. Dep. Agric. Nutr. Cent., Beltsville, Md.). *Fed. Proc.* 34(3):910, 1975.

1632 GLUCOSE TRANSPORT ACROSS THE SMALL INTESTINE OF PARENTERALLY NOURISHED RATS. *(E.)* Schanbacher, L. M.; Copeland, E. M.; Dudrick, S. J.; Johnson, L. R.; Castro, G. A. (Univ. Texas Med. Sch., Houston). *Fed. Proc.* 34(3):917, 1975.

1633 EFFECT OF ETHANOL (ETH) ON SODIUM DEPENDENT GLUCOSE (GL) TRANSPORT IN JEJUNUM. *(E.)* Beck, I. T.; Dinda, P. K.; Beck, M.; McElligott, T. F. (no affil.). *Fed. Proc.* 34(3):469, 1975.

1634 AN EFFECT OF SHORT CHAIN FATTY ACIDS ON TRANSEPITHELIAL PD OF RAT JEJUNUM. *(E.)* Baker, D.; Wall, Jr., M. J. (Univ. Texas Med. Branch, Galveston). *Fed. Proc.* 34(3):475, 1975.

1635 AN INFRADIAN ENTRAINMENT OF FAT AND PROTEIN ABSORPTION IN THE RAT. *(E.)* Squibb, Jr., R. E. (Dep. Food Sci., Rutgers Univ., New Brunswick, N.J.). *Fed. Proc.* 34(3):917, 1975.

1636 STUDIES OF ABSORPTION IN PROTEIN DEPLETION I. ABSORPTION OF NICOTINIC ACID, THIAMINE AND RIBOFLAVIN IN THE RAT. *(E.)* De Angelis, R.; Santi, G.; Rogano, R. N.; Vecchia, M. G. (Inst. Biomed. Sci., Sao Paulo Univ., Brazil). *Arq. Gastroenterol.* 11(4):223-228, 1974.

1637 THE EFFECT OF STRESS ON THE DIURNAL PATTERN OF INTESTINAL TRANSPORT OF ASCORBIC ACID IN THE GUINEA PIG. *(E.)* Litwornia, B. L. (no affil.). *Fed. Proc.* 34(3):917, 1975.

1638 VITAMIN D-DEPENDENT CALCIUM UPTAKE BY ISOLATED INTESTINAL CELLS. *(E.)* Freund, T. S.; Bronner, F. (Univ. Connecticut Health Cent., Farmington). *Fed. Proc.* 34(3):475, 1975.

1639 VITAMIN D_3-MEDIATED INTESTINAL CALCIUM ABSORPTIVE MECHANISM: REGULATION BY CYCLIC AMP AND CALCIUM. *(E.)* Corradino, R. A. (Dep. Phys. Biol., New York State Vet. Coll., Cornell Univ., Ithaca). *Fed. Proc.* 34(3):887, 1975.

1640 BASIC FEATURES OF THE VITAMIN D-DEPENDENT PHOSPHATE TRANSPORT BY CHICK JEJUNUM IN VITRO. *(E.)* Peterlik, M.; Wasserman, R. H. (Dep. Phys. Biol., Cornell Univ., Ithaca, N.Y.). *Fed. Proc.* 34(3):887, 1975.

1641 INTESTINAL CALCIUM AND SODIUM FLUXES IN CORTICOSTEROID-TREATED RATS. *(E.)* Adams, J. S.; Lukert, B. P. (Kansas Univ. Med. Cent., Kansas City). *Fed. Proc.* 34(3):917, 1975.

1642 EFFECT OF MAGNESIUM (Mg) ON RAT JEJUNAL SODIUM (Na) AND WATER (H_2O) TRANSPORT. *(E.)* DiBona, G. F.; Sawin, L. L. (Univ. Iowa Coll. Med., Iowa City). *Fed. Proc.* 34(3):469, 1975.

1643 THE EFFECT OF FRUSEMIDE ON SMALL-INTESTINAL ABSORPTION OF WATER AND ELECTROLYTES. *(E.)* Mackenzie, J. F.; Cochran, K. M.; Russell, R. I. (R. Infirm., Glasgow, Scotland). *Gut* 14(10):831, 1974.

1644 ION TRANSPORT CHARACTERISTICS OF HUMAN JEJUNAL AND ILEAL MUCOSA IN VITRO. *(E.)* Isaacs, P.; Turnberg, L. A. (Manchester R. Infirm., England). *Gut* 15(10):830–831, 1974.

1645 EFFECTS OF SALINE INFUSION ON THE INTENSITY OF INTESTINAL ABSORPTIVE PRESSURE IN THE RAT. *(E.)* Bresler, E. H.; O'Dell, R. M.; Nielsen, K. T.; Cazenavette, L. L. (VA Hosp., New Orleans, La.). *Fed. Proc.* 34(3):469, 1975.

1646 THE EFFECT OF PHARMACOLOGICAL DOSES OF PREDNISOLONE ON THE ABSORPTIVE AND DIGESTIVE FUNCTIONS OF THE RAT SMALL INTESTINE. *(E.)* Batt, R. M.; Peters, T. J. (R. Postgrad. Med. Sch., London, England). *J. Physiol. (Lond.)* 245(2):124P–125P, 1975.

1647 PARTIAL PURIFICATION AND PROPERTIES OF INTRINSIC FACTOR-BINDING RECEPTOR FROM GUINEA PIG ILEUM. *(E.)* Cotter, R.; Rothenberg, S. P. (New York Med. Coll., N.Y.). *Fed. Proc.* 34(3):475, 1975.

1648 POTASSIUM INFLUX ACROSS THE BASO-LATERAL MEMBRANES OF RABBIT ILEUM. *(E.)* Nellans, H. N.; Schultz, S. G. (Univ. Pittsburgh Sch. Med., Pa.). *Fed. Proc.* 34(3):285, 1975.

1649 ION TRANSPORT BY RABBIT COLON: EFFECT OF AMILORIDE. *(E.)* Frizzell, R. A.; Koch, M. J.; Cooper, D. H.; Schultz, S. G. (Univ. Pittsburgh Sch. Med., Pa.). *Fed. Proc.* 34(3):285, 1975.

1650 A STUDY OF ABSORPTION OF COMPOUNDS FROM THE RAT BILIARY TREE BY RETROGRADE INTRABILIARY INJECTION (RII). *(E.)* Peterson, R. E. (Med. Coll. Wisconsin, Milwaukee). *Fed. Proc.* 34(3):711, 1975.

1651 BILIARY EXCRETION OF IOPANOATE GLUCURONIDE IN RAT. *(E.)* Cooke, W. J.; Munck, L. (Univ. Massachusetts Med. Sch., Worcester). *Fed. Proc.* 34(3):711, 1975.

1652 HEPATIC TRANSMEMBRANE POTENTIALS IN HEMORRHAGIC SHOCK. *(E.)* Sayeed, M. M.; Adler, R.; D'Agrosa, L. S.; Chaudry, I. H.; Baue, A. E. (Jewish Hosp., St. Louis, Mo.). *Fed. Proc.* 34(3):379, 1975.

1653 DIMINISHED URINARY DIGOXIN (D) EXCRETION AFTER SALICYLAZOSULFAPYRIDINE (SASP). *(E.)* Juhl, R.; Summers, R. W.; Blaug, S.; Guillory, J. K. (Univ. Iowa Coll. Med., Iowa City). *Fed. Proc.* 34(3):916, 1975.

See also, 1615, 1766, 1781, 1830, 1832, 1840.

1654 THE EFFECT OF GLUCAGON AND THE COMBINED EFFECT OF GLUCAGON AND SECRETIN ON LOWER ESOPHAGEAL SPHINCTER PRESSURE IN MAN. *(E.)* Christiansen, J.; Borgeskov, S. (Bispebjerg Hosp., Copenhagen, Denmark). *Scand. J. Gastroenterol.* 9(7):615-618, 1974.

The effects of glucagon and glucagon plus secretin on resting and pentagastrin-induced lower esophageal sphincter pressure were studied in 15 healthy volunteers (5 females and 10 males, aged 20-28 yr). The registration of intraluminal pressures in the stomach, lower esophageal sphincter, and esophagus was obtained using 3 water-filled polyvinyl tubes connected to external transducers. Glucagon (20 µg/kg) was administered i.v. as a single 30-sec injection; pentagastrin (0.5 µg/kg/hr) was administered i.v. continuously throughout the study, beginning 45 min before the glucagon injection. Other subjects were given glucagon (20 µg/kg) and/or secretin (1 U/kg) i.v. as a single 30-sec injection. Pentagastrin caused a 29% increase in the mean resting lower esophageal sphincter pressure (17-19 mm Hg), while glucagon decreased the mean resting pressure by 58% and the mean pentagastrin-stimulated pressure by 50%. Secretin decreased the pentagastrin-stimulated sphincter pressure by 41%, and glucagon combined with secretin decreased it by 56%. Thus the effects of glucagon and secretin when combined were neither synergistic nor antagonistic. The data indicate that glucagon is a potent antagonist to the effect of gastrin on the lower esophageal sphincter; it is likely that this antagonism is competitive.

1655 RESTING PRESSURES IN THE LOWER ESOPHAGEAL SPHINCTER. *(E.)* Bloom, A. A.; Stekelman, M.; Varadee, R.; Carvajal, S.; Davidson, M. (Bronx-Lebanon Hosp. Cent., Bronx, N.Y.). *Am. J. Dig. Dis.* 19(12):1120-1123, 1974.

Interdeglutitive pressures of the lower esophageal sphincter were studied by nonpullthrough techniques in 36 untrained volunteers. With the subject fasting and recumbent, a tube assembly was passed p.o. so that at least 1 opening was in the lower esophageal sphincter. The stomach was perfused with 300 ml of 0.9 N saline or 300 ml of 0.1 N HCl without moving the tube assembly. The initial unstimulated sphincter pressures averaged 13.3 mm Hg above the pressures in the gastric fundus. In 30 subjects, a gradual and protracted fall in the baseline occurred beginning 2-15 min after entry into the sphincter; its magnitude averaged 12.8 mm Hg. Lower esophageal sphincter pressure could, but did not necessarily, decay uniformly throughout the entire length of the esophagus; decay in sphincter pressure was never accompanied by a rise in pressure at the adjacent gastric or esophageal orifices. Decay usually resulted in a basal pressure intermediate between the initial sphincter pressures and the gastrofundal pressure. Protracted decay did not correct itself spontaneously, but could be reversed by swallowing, movement of the assembly (as in a pullthrough procedure), or by infusion with NaCl solution. Perfusion of the fundus with HCl restored sphincter pressure in only 1 of 6 cases. The data indicate that under the conditions of this study, the lower esophageal sphincter does not present a continuous barrier of high pressure separating the stomach and esophagus; they also suggest a need to redefine the lower esophageal sphincter.

1656 INDEPENDENCE OF GASTRIN AND HISTAMINE RECEPTORS IN THE LOWER OESOPHAGEAL SPHINCTER OF THE MONKEY AND POSSUM. *(E.)* de Carle, D. J.; Glover, W. E. (Sch. Med., Univ. New South Wales, Australia). *J. Physiol. (Lond.)* 245(2):78P-79P, 1975.

The action of pentagastrin (6 µg/kg/hr continuous i.v. infusion or 0.5-5.0 µg/kg i.v. bolus) on lower esophageal sphincter pressure was studied in the monkey (*Macaca nemestrinae*) and the Australian brush-tailed opussum (*Trichosurus vulpecula*), two animals with sphincters resembling that of man both histologically and functionally. Pentagastrin alone by continuous infusion or bolus produced a 41-92% increase in lower esophageal sphincter pressure in opossums and a 113-197% increase in monkeys. Simultaneous infusion of metiamide (2.7-4.5 mg/kg/hr) and/or mepyramine (1.0-2.0 mg/kg/hr) had little or no effect on this. Histamine (5-50 µg/kg) caused a rapid dose-related fall in sphincter pressure in all but one of 10 animals. Although metiamide and mepyramine alone were ineffective in counteracting the histamine effect, both together almost abolished histamine-induced sphincter relaxation. Metiamide and mepyramine together, but not alone, also greatly reduced the histamine-induced fall in arterial blood pressure, indicating that both H_1- and H_2-receptors are involved in the depressor component of the arterial pressure response to histamine in both species. It is furthermore concluded that histamine and its receptors are not involved in the sphincteric response to pentagastrin. It is suggested that administration of H_2-receptor-antagonists to peptic ulcer patients should not cause reflux.

1657 GASTRIN PENTAPEPTIDE DECREASES CANINE GASTRIC TRANSMURAL PRESSURE. *(E.)* Wilbur, B. G.; Kelly, K. A. (Mayo Clin., Rochester, Minn.). *Gastroenterology* 67(6):1139-1142, 1974.

The mechanism by which gastrin slows the gastric emptying of liquids was studied. In 4 conscious, fasted female mongrel dogs, the transmural pressure of the stomach was measured continuously for 75 min, while 154 mM NaCl (0.8 ml/min) was infused i.v. In similar experiments using the same dogs, gastrin pentapeptide (0.1 µg/kg/min) was added to the NaCl infusion between the 15th and 45th min of infusion. In another set of experiments, a flaccid plastic bag inserted into the stomach was distended with 500 ml of a 5.0% aqueous solution of sodium diatrizoate; the position of the distended bag in the stomach was observed fluoroscopically during NaCl infusion and then during NaCl plus gastrin pentapeptide infusion. The mean gastric transmural pressure of about 11 cm H_2O changed little when NaCl alone was infused. The intragastric plastic bag used to distend the stomach occupied the gastric fundus and corpus, but not the antrum, in all 4

dogs. Within 2 min after the start of gastrin pentapeptide infusion, a decrease in the mean transmural pressure and in the amplitude of phasic changes in pressure occurred in each case. The mean pressure remained low throughout the infusion of gastrin pentapeptide and the phasic changes were nearly abolished. Both returned to normal values within 30 min of the cessation of gastrin pentapeptide infusion. The infusion of gastrin pentapeptide did not alter the position of the plastic bag in the stomach. Thus, gastrin pentapeptide decreased gastric transmural pressure in the stomach. The rapid onset of its action suggests that it acts directly on structures in the gastric wall, but it may act indirectly by stimulating the gastric secretion of HCl.

1658 SPIKE-FREE ACTIVATION MECHANISM IN SMOOTH MUSCLE OF GUINEA-PIG STOMACH. *(E.)* Golenhofen, K.; Wegner, H. (Physiol. Inst. Univ. Marburg, Germany). *Pfluegers Arch.* 354(1):29-37, 1975.

Electrical and mechanical activity of circular muscle strips of guinea-pig stomach were studied to determine whether a spike-free activation mechanism could play a role in normal activation of stomach smooth muscle. Circular strips of stomach muscular wall were excised from the distal corpus/proximal antrum region of 31 guinea-pigs and mounted in a perfused glass capillary; electrical activity was measured extracellularly by platinum wire electrodes and recorded simultaneously with mechanical activity. Krebs solution with and without verapamil was used as the perfusion medium. Spontaneous activity consisting of phasic contractions combined with spike potentials was augmented by acetylcholine (5×10^{-8} - 10^{-7} g/ml) and suppressed by verapamil (5×10^{-6} - 2×10^{-5} M/liter). Acetylcholine applied 15 min after treatment with verapamil produced a spike-free tonic activation. Under normal conditions phasic contractions were superimposed on this tonic activation. The acetylcholine-induced activation, therefore, consisted of 2 different components, one of which could be selectively blocked by verapamil. Both components disappeared in calcium-free solution. In 5 additional experiments using *taenia coli* preparations, no spike-free tonic activation could be elicited with acetylcholine. It is concluded that 2 different calcium activation systems are responsible for 2 components of activation of stomach smooth muscle.

1659 VAGAL CONTROL OF GASTRIC ELECTRICAL CONTROL ACTIVITY AND MOTILITY. *(E.)* Sarna, S. K.; Daniel, E. E. (Div. Biomed. Eng., Univ. Alberta, Edmonton, Canada). *Gastroenterology* 68(2): 301-308, 1975.

The nerves of Latarjet and those going directly to the fundus and the body of the stomach were stimulated in dogs to determine the effect on gastric electrical control activity (ECA) and contractions. The lower limits of effective stimulation parameters were: pulse frequency 2 pulses/sec; pulse width 2 msec; pulse amplitude 0.3 ma or 5 V. Stimulation at or near the lower limits of stimulation parameters caused contractions with little effect on gastric ECA. At higher pulse repetition rates and amplitudes, vagal stimulation caused premature control potentials (PCP's), delayed control potentials (DCP's), and contractions throughout the electrically active region. The PCP's and DCP's in the antrum became phase-unlocked from those in the corpus. After 2 to 5 min of such vagal stimulation, PCP's and DCP's disappeared; gastric control waves became phase-locked at normal frequency and aborad phase lag. Contractions continued to occur. Atropine and hexamethonium blocked all effects of vagal stimulation. After gastric ECA became insensitive to vagal stimulation, PCP's could still be produced by intraarterial acetylcholine or dimethylphenylpiperazinium, or by an electrical stimulus applied to gastric muscle, and physostigmine could temporarily restore ECA sensitivity to vagal stimulation. The ECA-insensitive state to vagal stimulation represents depletion of acetylcholine at preganglionic sites. It is concluded that since stimulation of vagal branches to the fundus and proximal corpus did not cause contractions or disruption in ECA pattern, those branches do not supply cholinergic motor fibers to the electrically active region of the stomach.

1660 GASTROINTESTINAL TRANSIT ESTIMATED BY AN ISOTOPE CAPSULE. *(E.)* Kirwan, W. O.; Smith, A. N. (West. Gen. Hosp., Edinburgh, Scotland). *Scand. J. Gastroenterol.* 9(8):763-766, 1974.

The adequacy of an isotope-containing capsule to measure total gastrointestinal and colonic transit time was evaluated by comparing it to the use of radio-opaque markers. After the capsule of perspex, containing approximately 10 µCi I^{131}, was swallowed, its exact position was determined using a sodium iodide scintillation counter. The capsule transit time was compared with that of radio-opaque markers in normal subjects and in patients with diarrhea and constipation. Stools were collected until at least 80% of the radio-opaque markers as well as the isotope capsule were recovered. In 10 normal subjects the transit time was 23 hr; in 5 constipated patients it was 187 hr. There was delay at the splenic flexure and rectosigmoid in constipated subjects but only at the rectosigmoid in normals. A high degree of correlation was observed between the 2 methods in normal subjects and in those with constipation, but less in cases of diarrhea (transit time too rapid for accurate measurement). Transit time was accelerated by varying the specific gravity of the isotope capsule from 1.1 (approximate specific gravity of gastrointestinal contents) to 1.35 or to 0.93. The acceleration was attributed to the possible separation of the heavier or lighter capsule from the luminal contents and its moving closer to the bowel wall, thus being more effectively propelled and excreted. It is concluded that an isotope capsule is an accurate tool (as reliable as radio-opaque markers) to monitor total, colonic or regional transit times in normal or constipated individuals.

1661 DUODENAL CONTROL OF GASTRIC EMPTYING IN THE MILK-FED CALF. *(E.)* Bell, F. R.; Mostaghni,

K. (Dep. Med., R. Vet. Coll., London, England). *J. Physiol. (Lond.)* 245(2):387-407, 1975.

The mechanisms of gastric (abomasal) emptying were studied in milk fed calves with open duodenal re-entrant cannulas which received duodenal infusions of various substances. Such a preparation allows for simultaneous infusion of test substances into the duodenum and collection of effluent fluid from the abomasum without mixing of the two. Sodium bicarbonate (200-500 mOsm/liter) or sodium chloride (200-300 mOsm/liter) infusion induced rapid abomasal emptying of a standard water test meal. Sodium bicarbonate was the more effective agent at all concentrations tested. Potassium chloride (100-700 mOsm/liter), calcium chloride (100-800 mOsm/liter), glucose (100-1200 mOsm/liter) and HCl (10-60 mEq/liter) all inhibited gastric emptying. Although hypotonic ammonium chloride (100-200 mOsm/liter) stimulated gastric emptying, isotonic or hypertonic ammonium chloride as well as urea, lactose and acetic acid had little effect. Hypertonic solutions of all test substances inhibited gastric emptying to varying degrees following duodenal instillation. The inhibitory and stimulatory effects of the test substances were independent of the abomasal contents. These results support the previously published view that osmotic control of gastric emptying resides in osmoreceptors within the duodenum.

1662 GASTRIC EMPTYING AND SECRETION OF BILE ACIDS, CHOLESTEROL, AND PANCREATIC ENZYMES DURING DIGESTION. DUODENAL PERFUSION STUDIES IN HEALTHY SUBJECTS. *(E.)* Brunner, H.; Northfield, T. C.; Hofmann, A. F.; Go, V. L. W.; Summerskill, W. H. J. (Mayo Clin., Rochester, Minn.). *Mayo Clin. Proc.* 49(11):851-860, 1974.

Hourly gastric emptying and secretion of bile acids, biliary cholesterol, and pancreatic enzymes were measured over a 12- or 24-hr period by a duodenal perfusion technique in 18 healthy persons receiving liquid test meals at 8:00 am, noon and 6:00 pm. Test meals varying in caloric content (20 cal/kg, low calorie; 30 cal/kg, medium calorie; and 40 cal/kg, high calorie) but identical in caloric distribution were infused into the stomach. Replicate studies established methodologic and biologic reproducibility. Gastric emptying, assessed by duodenal recovery of the meal marker, was exponential and related to meal size. Bile acid and pancreatic enzyme outputs were similar in amount with all meals during the day. At night, they decreased to basal levels more rapidly with the low calorie meal, as gastric emptying became complete. Total daily bile acid output was approximately 240 mM/kg for the low calorie meal and 420 mM/kg for the medium calorie and high calorie meals. Recycling of the bile acid pool averaged 4/day for the low calorie meal and 6/day for the high calorie meal. Daily cholesterol secretion, which correlated highly with bile acid secretion, was approximately 28 μM/kg for the low calorie meal and 42 μM/kg for medium calorie and high calorie meals. The type of the meal appeared to determine the duration of gastric emptying, which in turn influenced the duration of pancreatic and biliary secretions. A major determinant of total outputs in these studies may be a meal flow sufficiently great to evoke cholecystokinin-pancreozymin release from the small intestine.

1663 VOMITING: RADIOGRAPHIC AND OSCILLOGRAPHIC CORRELATES IN THE DECEREBRATE CAT. *(E.)* McCarthy, L. E.; Borison, H. L.; Spiegel, P. K.; Friedlander, R. M. (Dartmouth Med. Sch., Hanover, N.H.). *Gastroenterology* 67(6):1126-1130, 1974.

The visceral events associated with the mechanics of vomiting were observed radiographically in order to establish direct temporal correlations. Vomiting was evoked in 7 decerebrated, unanesthetized cats by i.m. administration of alkavervin (Veriloid) (50-100 g/kg). A barium meal (50-100 ml) was provided by stomach tube. The sequence of responses was followed cineradiographically using an image intensifier and appropriate filming procedures. Thoracic and abdominal venous pressures and diaphragmatic EMG's were monitored and recorded. The vomiting mechanism could be divided into 2 separate processes: retching and expulsion. Retching was characterized by repeated negative thoracic pressure pulses accompanied by diaphragmatic EMG activity (rapid descent of the diaphragm with concomitant positive pressure pulses in the abdomen). Contrast material from stomach moved to and fro into a dilated esophagus. Expulsion immediately followed the last retch and was characterized by a reversal of the thoracic pressure from negative to positive, continuing diaphragmatic EMG activity, sustained herniation of the gastric cardia into the thorax, and sudden cephalad movement of the diaphragm. These observations support the concept that retching is a preparatory maneuver in which the inherent antireflex characteristics of the abdominal esophagus and cardia are abolished by respiratory mechanisms.

1664 TWO DESCENDING NERVE PATHWAYS ACTIVATED BY DISTENSION OF GUINEA-PIG SMALL INTESTINE. *(E.)* Hirst, G. D. S.; Holman, M. E.; McKirdy, H. C. (Dep. Physiol., Monash Univ., Clayton, Australia). *J. Physiol. (Lond.)* 244(1):113-127, 1974.

The discharge of excitatory synaptic potentials produced in myenteric neurons by distension of an orally situated segment of intestine was studied. Intracellular recordings were made from myenteric neurons lying in a flap of plexus attached to the aboral end of a segment of mid-small intestine in the guinea pig. The preparations were bathed in modified Krebs solution containing atropine sulfate, tubocurarine chloride, or tetrodotoxin. Excitatory synaptic potential could be evoked in most neurons by distension of the attached intestinal segment; 2 distinct descending polysynaptic pathways could be activated in this way. Transient, short-latency (0.5 sec) discharges were recorded from neurons lying along 1 pathway, while the excitatory synaptic potentials from neurons on the other pathway were recorded intermittently during maintained distension with latencies of 2-11 sec. When intracellular recordings were made from circular muscle cells on the anal side, distension evoked a transient hyperpolarization after a latency of about 1

sec; this was followed by a more prolonged excitation of both circular and longitudinal muscles. It is suggested that 1 pathway is a descending inhibitory pathway, while the other is a descending excitatory pathway. Both pathways could be activated by brief or prolonged distension of the adjacent intestinal segment. There was a long latency before the onset of a synaptic discharge in the neurons on the excitatory pathways and the discharge of excitatory synaptic potentials persisted several sec after the stimulus had been removed. The descending inhibitory pathway may be 'gated' in some way; AH cells (cells in which action potentials are followed by prolonged afterhyperpolarization) may provide afferent input to this pathway and limit the firing of excitatory synaptic potentials along it. In the presence of a stimulus (e.g., a bolus), activation of the inhibitory pathway would cause hyperpolarization of the circular muscle layer; then, after a delay, interneurons and excitatory neurons influencing both the circular and longitudinal muscle would be discharged causing a wave of excitation to sweep in an aboral direction along the intestine. These sequences would recur along the intestine until the bolus had in some way been dissipated.

1665 ACTION OF SOME ANTISPASMODICS ON GASTROINTESTINAL MUSCLE. *(E.)* Benzi, G.; Arrigoni, E.; Panceri, P.; Villa, R. F.; D'Angelo, L.; Panzarasa, R.; Autelli, M.; Berte, F. (Dep. Pharmacol., Univ. Pavia, Italy). *Digestion* 11(1/2): 72-89, 1974.

1666 SLEEP AND ESOPHAGEAL MOTILITY. *(Ger.)* Stacher, G.; Fink, G. (1st Surg. Clin., Univ. Vienna, Austria). *Z. Gastroenterol.* 12(4): 251-256, 1974.

1667 LOWER-OESOPHAGEAL-SPHINCTER RELAXATION IS A MYTH. *(E.)* Sullivan, S. N. (Victoria Hosp., London, Canada). *Lancet* 2(7884):809-810, 1974.

1668 VAGAL MECHANORECEPTORS OF THE LOWER OESOPHAGEAL SPHINCTER AND OF THE PYLORIC SPHINCTER IN THE CAT. *(E.)* Bitar, K.; Mei, N.; Michelucci, M. H. (Inst. Neurophysiol. Psychophysiol., Marseille, France). *J. Physiol. (Lond.)* 245(2):103P-104P, 1975.

1669 TENSION RECEPTORS IN THE OESOPHAGUS AND STOMACH OF THE RAT. *(E.)* Clarke, G. D.; Davison, J. S. (Dep. Physiol., Univ. Dundee, Scotland). *J. Physiol. (Lond.)* 244(1):41P-42P, 1975.

1670 A METHOD OF DISPLAYING THE ELECTRICAL ACTIVITY OF CANINE GASTRIC MUSCULATURE. *(E.)* Levien, L. J. (Dep. Surg., Univ. Witwatersrand, Johannesburg, South Africa). *S. Afr. J. Surg.* 12(2):72, 1974.

1671 THE EFFECT OF METOCLOPRAMIDE ON THE GASTRIC EMPTYING OF WATER AND A SOLUTION OF 10% GLUCOSE. *(Sp.)* Rodrigo Gomez, J. M.; Alapont Raga, V.; Guix Garcia, J.; Aparisi Quereda, L.; Serra Desfilis, M. A.; Palao Esteve, J.; Garcia-Conde Gomez, F. J. (Fac. Med. Valencia, Spain). *Rev. Esp. Enferm. Apar. Dig.* 44(5):469-484, 1974.

1672 USE OF INTRAGASTRIC FRUCTOSE TOLERANCE TEST IN ASSESSMENT OF GASTRIC EMPTYING. *(E.)* Pettit, G. W.; Bostian, K. A.; Elwell, M. R. (USA Med. Res. Inst. Infect. Dis., Fort Detrick, Md.). *Fed. Proc.* 34(3):910, 1975.

1673 SYNAPTIC POTENTIALS RECORDED FROM SOME NEURONES OF THE SUBMUCOUS PLEXUS OF GUINEA-PIG INTESTINE. *(E.)* Hirst, G. D. S.; McKirdy, H. C. (Dep. Physiol., Monash Univ., Clayton, Australia). *J. Physiol. (Lond.)* 244(1):35P-36P, 1975.

1674 EFFECTS OF PAPAVERINE ON THE RABBIT ILEUM UNDER THE INFLUENCE OF ALTERED TEMPERATURE AND EXTRACELLULAR Ca^{2+}. *(E.)* Reinhardt, D.; Wagner, J.; Schumann, H. J. (Inst. Pharmacol., Univ. Essen, Germany). *Eur. J. Pharmacol.* 29(2):279-283, 1974.

1675 INTESTINAL SMOOTH MUSCLE RESPONSE TO CHRONIC OBSTRUCTION: POSSIBLE APPLICATIONS IN JEJUNOILEAL ATRESIA. *(E.)* Cloutier, R. (Univ. Laval Hosp. Cent., Quebec, Canada). *J. Pediatr. Surg.* 10(1):3-8, 1975.

1676 INTESTINAL MYOELECTRIC ACTIVITY IN RESPONSE TO CHOLERA. *(E.)* Mathias, J. R.; Carlson, G. M.; Dimarino, A. J.; Bertiger, G.; Cohen, S. (Dep. Med., Univ. Pennsylvania, Philadelphia). *Fed. Proc.* 34(3):423, 1975.

1677 EFFECT OF PENTAGASTRIN ON ELECTRICAL ACTIVITY OF THE SMALL INTESTINE. *(E.)* Weisbrodt, N. W.; Copeland, E. M.; Kearley, R. W.; Moore, E. P.; Johnson, L. R. (Univ. Texas Med. Sch., Houston). *Tex. Rep. Biol. Med.* 32(2):586, 1974.

1678 MOTILITY OF THE LARGE INTESTINE OF MICE WITH HEREDITARY MEGACOLON. *(E.)* Brann, L.; Wood, J. D. (Univ. Kansas Med. Cent., Kansas City). *Fed. Proc.* 34(3):423, 1975.

1679 A PHARMACOLOGICAL ANALYSIS OF THE INHIBITORY EFFECTS OF THE SYMPATHETIC NERVES ON THE RABBIT COLON. *(E.)* Gillespie, J. S.; Khoyi, M. A. (Dep. Pharmacol., Univ. Glasgow, Scotland). *J. Physiol. (Lond.)* 244(1):42P-43P, 1975.

1680 SACRAL PARASYMPATHETIC REFLEX PATHWAY TO THE LARGE INTESTINE OF THE CAT. *(E.)* Krier, J.; de Groat, W. C. (no affil.). *Fed. Proc.* 34(3):423, 1975.

1681 RESPONSES OF NEURONS IN THE INFERIOR MESENTERIC GANGLION TO REPETITIVE STIMULATION OF PREGANGLIONIC INPUTS. *(E.)* Weems, W. A.; Szurszewski, J. H. (Mayo Found., Rochester, Minn.). *Fed. Proc.* 34(3):423, 1975.

1682 HORMONAL CONTROL OF COLONIC MYOELECTRICAL ACTIVITY. THE EFFECT OF PENTAGASTRIN AND GLUCAGON. *(E.)* Taylor, I.; Smallwood, R.; Brown, B. H.; Duthie, H. L. (no affil.). *S. Afr. J. Surg.* 12(2):72, 1974.

1683 EFFECTS OF BILE ACIDS ON THE MOTILITY OF THE COLON. *(E.)* Kirwan, W. O.; Smith, A. N.; Mitchell, W. D.; Eastwood, M. A. (West. Gen. Hosp., Edinburgh, Scotland). *Gut* 15(10):828, 1974.

1684 THE CHARACTER OF THE CAPACITANCE VESSEL RESPONSES IN THE SPLEEN AND INTESTINE UNDER ELECTRICAL STIMULATION OF THE SYMPATHETIC FIBRES. *(E.)* Tkachenko, B. I.; Medvedeva, N. J.; Pozdnjakov, P. K. (Inst. Exp. Med., Leningrad, USSR). *Experientia* 30(12):1413-1414, 1974.

1685 FUNCTIONAL DESCRIPTION OF THE CANINE CHOLEDOCHODUODENAL FLUTTER VALVE. *(E.)* Kendall, F. M.; Innes, D. L.; Martin, J. S.; Tansy, M. F. (Temple Univ. Health Sci. Cent., Philadelphia). *Fed. Proc.* 34(3):422, 1975.

1686 CONTINUOUS POSITIVE-PRESSURE VENTILATION AND CHOLEDOCHODUODENAL FLOW RESISTANCE. *(E.)* Johnson, E. E.; Hedley-Whyte, J. (Harvard Med. Sch., Boston, Mass.). *Fed. Proc.* 34(3):394, 1975.

1687 NEUROHUMORAL MOTORICITY OF THE CANINE EXTRAMURAL COMMON BILE DUCT. *(E.)* Murphy, J. J.; Tansy, M. F. (Temple Univ. Health Sci. Cent., Philadelphia, Pa.). *Fed. Proc.* 34(3):422, 1975.

1688 PRESSURE-VOLUME CHARACTERISTICS OF THE GALLBLADDER. *(E.)* Ryan, J.; Cohen, S. (Hosp. Univ. Pennsylvania, Philadelphia). *Fed. Proc.* 34(3):422, 1975.

See also, 1924, 1925, 1926.

SECRETION AND METABOLISM

1689 ROLE OF CALCIUM AND CYCLIC NUCLEOTIDES IN α-AMYLASE RELEASE FROM SLICES OF RAT PAROTID: STUDIES WITH THE DIVALENT CATION IONOPHORE A-23187. *(E.)* Butcher, F. R. (no affil.). *Fed. Proc.* 34(3):453, 1975.

1690 STUDIES ON Ca^{2+}-STIMULATED ADENOSINE TRIPHOSPHATASE IN RAT SUBMANDIBULAR GLANDS: ITS DISTRIBUTION AND PROPERTIES. *(E.)* Furuyama, S.; Negishi, T.; Tobe, Y.; Takiguchi, H. (Nihon Univ. Sch. Dent., Matsudo, Japan). *J. Dent. Res.* 54(1): 32-40, 1975.

1691 UNEXPECTED β ADRENERGIC EFFECTS OF THE ANTI-TUMOR AGENT, CYCLOCYTIDINE, ON RAT SALIVARY GLANDS. *(E.)* Schneyer, C. A.; Galbraith, W. M.; Mellett, L. B. (Univ. Alabama Med. Cent., Birmingham). *Fed. Proc.* 34(3):453, 1975.

1692 STUDIES ON ENZYME SECRETION AND CYCLIC AMP IN THE SUBMAXILLARY GLAND AND PANCREAS. *(E.)* Bhoola, K. D.; Lemon, M. J. C. (Med. Sch., Bristol, England). *J. Physiol. (Lond.)* 245(2):121P-122P, 1975.

See also, 1604, 1662.

SECRETION AND METABOLISM

Stomach

1693 INHIBITION OF IN VITRO STIMULATED GASTRIC ACID SECRETION BY A HISTAMINE H_2-RECEPTOR ANTAGONIST, METIAMIDE. *(E.)* Wan, B. Y. C.; Assem, E. S. K.; Schild, H. O. (Dep. Pharmacol., Univ. Coll. London, England). *Eur. J. Pharmacol.* 29(1):83-88, 1974.

The effect of a specific histamine H_2-receptor anta-

gonist, metiamide, on histamine- or pentagastrin-induced gastric acid secretion was studied in the isolated frog gastric mucosa and the isolated rat stomach. Histamine (10^{-5} M) treatment of the frog gastric mucosa increased acid secretion from control level (5.03 μEq H^+/cm^2/30 min) to 10.62 μEq H^+/cm^2/30 min; pentagastrin (4 x 10^{-6} M) treatment increased acid secretion to 11.14 μEq H^+/cm^2/30 min. Metiamide (10^{-4} M) administered together with histamine reduced the histamine effect by 1/2 (7.42 μEq H^+/cm^2/30 min); metiamide together with pentagastrin completely abolished the pentagastrin effect (5.31 μEq H^+/cm^2/30 min). Consistent and significant stimulation of acid secretion by histamine and pentagastrin could be obtained in isolated rat stomach providing the preparations were selected for low basal secretion rates (less than 6 μEq H^+/cm^2/30 min), and the concentrations of stimulants be within their effective range (12-50 μg histamine/ml or 0.2-1.0 μg pentagastrin/ml). Metiamide (10 μg/ml) produced significant inhibition of stimulation at all concentrations of histamine and a persistent inhibition (up to 4 hr) of pentagastrin stimulation. These results suggest that the H_2-receptor antagonist, metiamide, acts directly on the acid secretory mechanisms of the gastric mucosa, although these *in vitro* findings do not rule out additional *in vivo* effects such as alterations in gastric mucosal blood flow.

1694 EFFECT OF BURIMAMIDE AND METIAMIDE ON PENTAGASTRIN-STIMULATED GASTRIC ACID SECRETION AND GASTRIC MUCOSAL BLOOD FLOW IN CATS. *(E.)* Albinus, M.; Sewing, K. F. (Dep. Pharmacol., Univ. Tubingen, Germany). *Experientia* 30(12):1435-1437, 1974.

The effects of 2 histamine-H_2-receptor-antagonists, burimamide and metiamide, on inhibition of pentagastrin-induced gastric acid secretion and gastric mucosal blood flow were compared in fasted, anesthetized male and female cats provided with acute gastric fistulas. Gastric secretory volume in response to pentagastrin (0.2 μg/kg/min i.v.) was characterized by a rapid rise to a maximum of 11 ml/30 min followed by a gradual decrease to 5 ml/30 min after 2 hr. Release of H^+ and Cl^- paralleled this pattern, reaching maxima of 145 mEq/liter and 170 mEq/liter, resp. Although burimamide (4 mg/kg i.v.) had no effect on any of these parameters, metiamide (2.9 mg/kg), when injected 60-90 min after the start of pentagastrin infusion, produced a significant decrease in gastric secretion (50%), acid secretion (56%) and mucosal blood flow (about 40%), but had no effect on Cl^- secretion. The failure of burimamide to have an effect is not in agreement with results of previous studies which utilized higher concentrations. The results with metiamide in which gastric secretion is inhibited to a greater extent than mucosal blood flow indicate that pentagastrin-induced secretion is more sensitive to H_2-receptor blockade.

1695 ELUCIDATION BY A H_2-RECEPTOR ANTAGONIST OF THE SIGNIFICANCE OF MUCOSAL HISTAMINE MOBILIZATION IN EXCITING ACID SECRETION. *(E.)* Lundell, L. Inst. Physiol., Univ. Lund, Sweden). *J. Physiol. (Lond.)* 244(2):365-383, 1974.

The effect of H_2-receptor blockade with metiamide on hormonal- or cholinergically-induced gastric acid secretion was studied in fasted female Sprague-Dawley rats with Heidenhain or Pavlov pouches with or without antral resection. Metiamide (4.9 mg/kg/hr i.v.) produced a 3-fold reduction in the maximum rate of gastric acid secretion (93.6 μEq/30 min) induced by pentagastrin (1 μg/hr i.v.), but had no effect on urinary histamine excretion (about 25 μg/hr). Mucosal histamine content was greater and formation was less in metiamide (9.7 mg/kg/hr) plus pentagastrin infused rats than in rats treated only with pentagastrin. Metiamide infusion also blocked methacholine-induced (10 μg/hr) acid secretion, acid secretion secondary to vagal stimulation with 2-deoxy-D-glucose (50 mg/kg with or without antral resection), and acid secretion occurring in response to feeding in animals with Pavlov pouches with or without antral resection. Metiamide had no effect on acid secretion in methacholine-treated animals also treated with pentagastrin, nor did it affect pepsin secretion in response to cholinergic stimulation. Metiamide (9.7 mg/kg/hr) inhibited the increase in gastric acid secretion in response to cholecystokinin-pancreozymin (1 μg/kg/hr) infusion. These results show that all stimuli known to mobilize histamine from gastric mucosa concurrently elicit acid secretory response which can be inhibited by H_2-receptor blockade. They also re-emphasize the importance of gastric mucosal histamine in stimulating parietal cell activity.

1696 HISTAMINE, PENTAGASTRIN, METHYL XANTHINES AND ADENYL CYCLASE ACTIVITY IN ACID SECRETION. *(E.)* Cooke, A. R.; Chvasta, T. E.; Granner, D. K. (Univ. Hosp., Univ. Iowa, Iowa City). *Proc. Soc. Exp. Biol. Med.* 147(3):674-678, 1974.

The possible role of pentagastrin in the stimulation of gastric acid secretion *via* cyclic AMP was tested by studying the *in vivo* effects of the methyl xanthines on acid output stimulated by histamine or pentagastrin in 16 male volunteers and in 4 dogs with gastric fistulas and Heidenhain pouches. The amounts of the substances infused in the dogs for 2 hr following determination of basal secretion levels were: caffeine (base) 10, 20, and 40 mg/kg/hr; aminophylline 5, 10, and 20 mg/kg/hr; pentagastrin 0.5, 1, 2, 4, and 8 μg/kg/hr, with or without caffeine, 10 mg/kg/hr; histamine dihydrochloride 0.0125, 0.025, 0.05, 0.1, 0.2, and 0.4 mg/kg/hr with or without caffeine 10 mg/kg/hr; and pentagastrin 1, 2, and 4 μg/kg/hr with 10 or 20 mg/kg/hr aminophylline. Gastric juice was collected from the gastric fistula and Heidenhain pouch every 15 min. Of the 16 human subjects (8 of whom were healthy students, and the rest suffering from either gastric or duodenal ulcer disease), 6 received caffeine in doses of 3 and 6 mg/kg/hr by i.v. infusion, the dose being doubled after 60 min. In another 5 subjects, 6 μg/kg/hr pentagastrin with or without caffeine (4 mg/kg/hr) was infused i.v. for 120 min; and in the last 5 subjects, histamine acid phosphate (0.04 mg/kg/hr) with or without caffeine (4 mg/kg/hr) was infused i.v. for 120 min. Gastric juice was collected at 10-min intervals. Pentagastrin-stimulated acid output was not increased by the methyl xanthines in dogs or man. Acid output in response to histamine was potentiated by caffeine

in the dog but not in the human studies. Neither caffeine nor aminophylline when infused alone stimulated acid output in the dog. At the end of the animal experiments, studies were made *in vitro* of adenyl cyclase activity in fundic and antral mucosa. Adenyl cyclase activity in dog gastric mucosa was not stimulated by histamine or pentagastrin. Previous conflicting studies about the role of cyclic AMP in acid secretion are briefly discussed, and it is concluded that it is difficult to determine at present whether mammalian gastric secretion is related to, or independent of, changes in cyclic AMP.

1697 ACID SECRETORY RESPONSES TO HISTAMINE AND PENTAGASTRIN IN CONSCIOUS MONKEYS. *(E.)* Rosato, E. F.; Mayer, L. J.; Arenschield, S.; Rosato, F. E.; Brooks, F. P. (Univ. Pennsylvania Sch. Med., Philadelphia). *Am. J. Dig. Dis.* 19(12):1111-1115, 1974.

Acid secretory responses to i.v. pentagastrin and histamine were studied in 8 male rhesus monkeys equipped with a gastric cannula and an indwelling polyvinyl catheter in the inferior vena cava which were chronically restrained in primate chairs. Histamine acid phosphate was infused i.v. at a constant rate of 30 ml/hr in doses of 0.02, 0.04, and 0.08 mg/kg/hr histamine acid phosphate. Pentagastrin was infused at a rate of 30 ml/hr in doses of 1.5, 7.5, and 15 µg/kg/hr. The animals were studied during 41 experiments for histamine and 43 for pentagastrin. The maximal acid response to histamine ranged from 2.57-8.39 mEq/hr and occurred within 15-25 min of the initiation of the histamine infusion. The maximal acid response to pentagastrin varied from 2.09-6.89 mEq/hr. For both stimulants the dose response curves were similar to those described for man with respect to sensitivity to stimulus, onset of acid response, and the response to supramaximal stimulation. The maximum acid response to histamine was significantly greater than the response to pentagastrin in 3 monkeys. The reason for this individual variation in response to the 2 stimulants is not known. There was no evidence of fade of acid secretory response at any dose level when analyzed for individual collection periods over a 45-min experiment. The lack of demonstrable secretory fade, however, does not rule out the presence of tachyphylaxis because of the technique of stepwise increase in pentagastrin dosage during single experiments. The use of this technique does not allow for accurate control of the blood levels of pentagastrin, and it is possible that accumulation of high levels of pentagastrin occurs in certain individuals. In 1 monkey, however, several tests were run where only the 7.5 and 15 µg/kg/hr pentagastrin doses were used and no difference in acid secretion was noted. Since both of these doses were supramaximal for this animal, it is concluded that tachyphylaxis was not responsible for the decreased acid response when compared with histamine in this monkey.

1698 INTERDIGESTIVE AND HISTAMINE-STIMULATED GASTRIC SECRETION IN GUINEA PIGS BEARING PERMANENT GASTRIC AND VASCULAR CANNULAS. *(E.)* Kowalewski, K. (Surg. Med. Res. Inst., Univ. Alberta, Edmonton, Canada). *Digestion* 11(1/2):2-11, 1974.

Spontaneous interdigestive and histamine-stimulated gastric acid secretion were studied in fasted adult male guinea pigs supplied with permanent gastric and i.v. cannulas. Secretion with or without continous infusion of histamine (6.5-75 mg/kg/24 hr) was studied by collecting samples in 3, 3 and 18 hr fractions over a 24 hr period. Acid and pepsin secretion in unstimulated, as well as in animals infused with high doses (50-75 mg/kg/24 hr) of histamine, was highly variable, with some samples not containing detectable H^+. All samples, however, contained pepsin. In general, secretion volume, gastric acid output and concentration and pepsin secretion were higher than controls during the first 3 hr of histamine infusion with levels gradually decreasing thereafter. The maximally effective histamine dose which produced the greatest 24 hr output of acid (4.20 mg) and pepsin (212 mg) was 12.5 mg/kg/24 hr. This dose was about 8 times less than the maximally effective dose previously reported for rats. The fact that temporary achlorhydria has not been reported in rats suggests that vagal tonus may play a more important role in the interdigestive secretory phase in rats than in guinea pigs.

1699 ACID SECRETION AND MOBILIZATION OF GASTRIC MUCOSAL HISTAMINE ON COMBINED CHOLINERGIC AND PENTAGASTRIN STIMULATION. *(E.)* Lundell, L. (Inst. Physiol., Univ. Lund, Sweden). *Agents Actions* 4(4):211-216, 1974.

Gastric secretion and histamine excretion were studied in an investigation of sites of interaction between cholinergic stimuli, pentagastrin, and gastric mucosal histamine. Sprague-Dawley rats were provided with Pavlov pouches in the oxyntic gland area, and were fasted 18 hr before each experiment. 2-Deoxyglucose (50 mg/kg), methacholine (10 µg/hr), or pentagastrin (0.0625-4 µg/hr) were administered i.v. Combinations were also used: pentagastrin (.0625-4 g/hr) plus 2-deoxyglucose (50 mg/kg); methacholine (10 g/hr) plus pentagastrin (8 g/hr); or methacholine (10 g/hr) plus histamine (1000 µg/hr). Gastric juice was collected by perfusion, and output determined, and the urine was assayed for histamine content. The vagal stimulant 2-deoxyglucose caused a rapid acid secretion rise to 92.5 µEq/30 min after 60 min, from an interdigestive level of 40.0 µEq/30 min. Histamine excretion was increased significantly from 12.5 µg/2 hr to 16.5 µg/2 hr. Even the lowest pentagastrin dose enhanced the effect of 2-deoxyglucose, and 0.25 µg/hr caused the maximal effect (337.4 µEq/2 hr), which was significantly higher than the effect of pentagastrin alone at 8 µg/hr (273.8 Eq/2 hr). A dose of 4 µg/hr of pentagastrin brought up histamine excretion to a maximum of 39.4 µg/2 hr. Methacholine alone caused no significant changes, but combined with pentagastrin (8 µg/hr), induced a secretion rate of 417 µEq/2 hr above interdigestive levels, significantly higher than that obtained from combined 2-deoxyglucose and pentagastrin stimulation (325.5 µEq/2 hr). The combined effect was not seen in histamine excretion. Methacholine and histamine combined caused a maximum secretion rate of 120 µEq/

30 min after 60 min. It is concluded that the vagal nerve has a facilitating influence on the parietal cell proper, rather than on the adjacent histamine-producing cells.

1700 THE EFFECT OF GRADED DOSES OF PENTAGASTRIN AND SECRETIN ON THE INTRINSIC FACTOR (IF) SECRETION IN MAN. *(E.)* Vatn, M. H.; Petersen, H.; Myren, J. (Ulleval Hosp., Oslo, Norway). *Scand. J. Gastroenterol.* 9(7):691-695, 1974.

The mode of intrinsic factor secretion was investigated using a wide dose range of pentagastrin and secretin in 3 series of experiments on healthy students of both sexes. In the 1st experiment, i.v. infusions of stepwise increasing doses of 0.078, 0.23, 0.7, and 2.1 U/kg/hr of secretin in physiologic saline solution were given at 37 ml/hr, each dose step lasting 30 min. In the 2nd experiment, i.v. infusions of stepwise increasing doses of 0.028, 0.167, 1.0, and 6.0 µg/kg/hr of pentagastrin in physiologic saline solution were given; and in the 3rd, i.v. infusions of 0.028, 0.167, 1.0, and 6.0 µg/kg/hr of pentagastrin were given on separate days with 1 week intervals to 5 individuals in randomized order. In experiment I, gastric and duodenal juice were collected by a Lagerlof tube in 10-min samples; in experiments II and III, 10-min samples of gastric juice were collected by a Levine tube. In experiment I, mean reflux of duodenal juice during stimulation in 9 examinations was 2.1 ml/30 min, range 0-12.25 ml. A reduction was found in the output of intrinsic factor, acid and volume of gastric juice by increasing doses of secretin. The output of pepsin was unchanged. No significant changes were found for concentrations of intrinsic factor and acid, whereas that of pepsin increased. In experiment II, a continuous 2-hr secretion of intrinsic factor on a high plateau level was demonstrated. An initial peak intrinsic factor output was observed on each dose level. The response was similar to that of pepsin, with a lower response to 6.0 than to 1.0 µg/kg/hr, in contrast to that of volume and acid. In experiment III, the responses to pentagastrin were higher after 6.0 µg/kg/hr for intrinsic factor and pepsin. Pepsin secretion continued on a high plateau level, compared to a low plateau level for intrinsic factor. It is suggested that the results support previous assumptions that secretion in the first 30 min contains preformed intrinsic factor. The increase of intrinsic factor secretion from basal to a low plateau level by even the smallest dose of pentagastrin may be the result of incomplete basal aspiration, continued secretion of preformed intrinsic factor, or stimulation of intrinsic factor synthesis. It is concluded that there is a dose-dependent secretion of intrinsic factor, pepsin, and acid to stimulation by 0.028 to 6.0 g/kg/hr of pentagastrin; the secretory mechanisms, however, appear to be different for the 3 substances.

1701 THE EFFECT OF CALCIUM AND PENTAGASTRIN ON THE GASTRIC SECRETION OF ACID, PEPSIN, AND INTRINSIC FACTOR (IF) IN MAN. *(E.)* Vatn, M. H.; Berstad, A.; Schrumpf, E. (Ulleval Hosp., Oslo, Norway). *Scand. J. Gastroenterol.* 9(7):697-701, 1974.

The effect of serum calcium on gastrin concentration, on acid, pepsin and intrinsic factor secretion, and on gastric secretion stimulated by different doses of pentagastrin was studied in 7 experiments carried out after an overnight fast in each of 5 healthy students of both sexes. Gastric secretion of acid, pepsin, and intrinsic factor, and serum gastrin concentrations were measured during i.v. infusions of calcium gluconate (4 mg Ca^{++}/kg/hr). No significant changes in serum gastrin concentrations were measured, in spite of an increase in mean serum calcium concentration from 9.7 to 11.8 mg/100 ml. The total outputs of pepsin and intrinsic factor increased significantly, whereas the increase of acid was not significant. Pentagastrin in doses of 0.1, 1.0, or 3.0 µg/kg/hr was given alone or combined with 4 mg/kg/hr Ca^{++} as calcium gluconate during 105 min. Calcium infusion significantly decreased acid secretion stimulated by the highest dose of pentagastrin. The intrinsic factor output stimulated by the lowest dose of pentagastrin was significantly increased by calcium. The increase of pepsin and intrinsic factor outputs is in agreement with the previously published concept of a stimulatory effect of calcium on macromolecular secretion. The results suggest that the effect of calcium on gastric secretion cannot be accounted for by changes in plasma gastrin concentration. There is no potentiation by calcium of pentagastrin-evoked secretion of acid, pepsin, or intrinsic factor, but a more complex interaction resulting in both stimulation and inhibition, depending on the dose of pentagastrin applied.

1702 FAILURE OF PENTAGASTRIN TO STIMULATE CYCLIC AMP ACCUMULATION IN HUMAN GASTRIC MUCOSA. *(E.)* Domschke, W.; Domschke, S.; Rosch, W.; Classen, M.; Demling, L. (Dep. Med., Univ. Erlangen-Nuremberg, Erlangen, Germany). *Scand. J. Gastroenterol.* 9(5): 467-471, 1974.

The effect of pentagastrin on the cyclic 3'5'-adenosine monophosphate (cyclic AMP) content of human gastric mucosa was studied in 21 healthy subjects. The cyclic AMP content of gastric mucosa, (measured by isotope dilution in mucosal biopsies taken from the corpus of the stomach during gastroscopy) did not change 30 min after pentagastrin administration (6 g/kg, s.c.), which is approximately the time of peak gastric acid output. Gastric acid secretion before and after pentagastrin administration did not correlate with cyclic AMP. In 2 patients with elevated serum gastrin and excessive basal hyperchlorhydria due to Zollinger-Ellison syndrome and insulinoma, resp., mucosal content of cyclic AMP was not different from the controls. However, previously reported compartmentalization of intracellular cyclic AMP, the hetereogeneity of biopsy material, and possible increased turnover rate of cyclic AMP in response to pentagastrin are factors which make the possible role of cyclic AMP in human gastric secretion unclear.

1703 DOSE-RESPONSE CURVE TO SECRETIN ON GASTRIC MUCUS SECRETION IN CONSCIOUS CATS. *(E.)* Vagne, M. (INSERM, Edouard Herriot Hosp., Lyon, France). *Digestion* 10(6):402-412, 1974.

The effect of secretin on gastric mucous secretion in the cat was studied to determine whether secretion was proportional to dosage, and whether its action persisted throughout an i.v. infusion. Three cats were given a Heidenhain pouch and a gastric fistula in the remaining part of the stomach, so that results could be evaluated with and without duodenal reflux. After an 18 hr fast, the gastric secretions from the gastric fistula, and the pouch were collected for 1 hr during a continuous i.v. infusion of 0.15 M saline at 13 ml/hr. Then secretin was added to the infusion at doses of 0.5, 3, and 18 U/kg/hr. For the first 2 dosages, collections were made after 3 hr periods; for the last, after one 2 hr period. Concentrations and outputs of pepsin, uronic acid, fucose, galactose, and total acid were determined. Determinations from the pouch indicated no change in acid output or concentration, while pepsin showed a significant increase at the dosages of 3 and 18 U/kg/hr. Fucose also showed a significant increase, as did the uronic acids and galactose, lasting throughout the infusion. Determinations from the fistula showed a decrease related to biliary reflux. Pepsin showed an increase at all doses, and the uronic acids were increased significantly. Fucose showed a weak increase due to bile reflux interference, and the galactose showed little change. The effects all lasted throughout infusion. It is concluded that the stimulant effect of secretin is proportional to the dose, and is greater on the polysaccharide than the glycoprotein fractions.

1704 INTERACTION OF DISULFIRAM AND ETHANOL ON GASTRIC MUCOSAL SECRETION AND BLOOD FLOW IN THE RAT. *(Fr.)* de Saint Blanquat, G.; Ozturkcan, O.; Derache, R. (Inst. Physiol., Paul Sabatier Univ., Toulouse, France). *Arch. Int. Pharmacodyn.* 208(2): 343-356, 1974.

Adult rats were given: (1) disulfiram (0-800 mg/kg) by gastric intubation; (2) ethanol (0.5 and 1 g/kg) by gastric intubation, and ethanol (0-4 g/kg) i.p.; and (3) both disulfiram and then ethanol, 2.5 hr later. In all cases, blood flow in the gastric mucosa was estimated by measuring the aminopyrine clearance along with the volume of secretion, free acidity, total acidity, and proteolytic activity. When the contents of the stomach were removed from rats treated with disulfiram alone, a whitish pellet of disulfiram was found; the mucosa appeared normal otherwise. After 2 hr, disulfiram significantly increased the volume, free acidity, total acidity, and proteolytic activity of the gastric juice, especially at moderate doses. The greatest increase in blood flow also occurred in these animals. There were significant positive correlations between blood flow and the secretory parameters. Administration of the larger dose of ethanol p.o. significantly increased the acidity, proteolytic activity, and blood flow above those in controls. There were also significant differences in the parameters for the 2 groups of controls, which differed only in the amount of liquid given. This result confirms the expectation that the direct action of alcohol on the gastric mucosa would distort the results. After i.p. administration of ethanol, there were significant deviations from control values in almost all cases, but not always in the same direction. There was a significant correlation between blood flow and secretion. Of the rats treated with both disulfiram and ethanol, all developed increased respiration and tachycardia less than 10 min after administration of ethanol; this lasted 20-30 min. The volume and proteolytic activity of the gastric juice did not change at all from control values; the free acidity and total acidity were consistently elevated; and blood flow increased following a large dose of disulfiram but was not affected by ethanol. There was a positive correlation between blood flow and volume and between free acidity and blood flow. The effects of ethanol on gastric secretion are consistent with those reported elsewhere for the rat and the dog. These findings indicate that the action of alcohol and disulfiram is neither cumulative nor antagonistic.

1705 DISSOCIATION OF GASTRIC ACID AND SERUM GASTRIN RESPONSES TO INTRALUMINAL CALCIUM IN MAN: INFLUENCE OF CALCITONIN AND PARATHYROID HORMONE. *(E.)* Holtermuller, K. H.; Goldsmith, R. S.; Sizemore, G. W.; Go, V. L. W. (Mayo Clin., Rochester, Minn.). *Gastroenterology* 67(6):1101-1106, 1974.

The effect of intragastric calcium on gastric acid secretion and serum gastrin in 19 healthy volunteers was characterized using an intragastric perfusion technique and its alteration by parathyroid hormone or calcitonin was investigated. Intragastric calcium chloride perfusion (80 mM, 5 ml/min) caused a 3-fold increase in gastric acid output without a change in serum gastrin and serum calcium values. The acid response to intragastric calcium represented a mean of 31.0% of the maximal gastric secretory capacity as determined with betazole. This effect was suppressed by calcitonin, but not influenced by parathyroid hormone, without a change in serum gastrin and serum calcium values. The mean calcium recoveries in gastric aspirate, expressed as percentage of calcium infused, were similar in these 3 experiments: calcium perfusion alone, 90.5%; calcium perfusion plus i.v. infusion of parathyroid hormone, 95.3%; and calcium perfusion plus i.v. infusion of calcitonin, 96.3%. There was no significant difference in the hourly gastric output of acid after perfusion of calcium chloride at pH 3.0 or pH 6.0. Perfusion of calcium into the duodenum, in an amount calculated to be the maximum leaked through the pylorus during the intragastric perfusion studies (12 mM, pH 6.0), did not cause gastric hypersecretion and gastrin release. In radioimmunoassay studies, the serum immunoreactive parathyroid hormone was normal ($\leq$ 40 µl-Eq/ml) in all subjects; plasma calcitonin remained undetectable (<50 pg/ml). These findings suggest that calcium can cause gastric hypersecretion by a direct effect on the parietal cells independent of the systemic calcium effect on gastric acid secretion.

1706 RELEASE OF GASTRIN AND STIMULATION OF ACID SECRETION BY BOMBESIN IN DOG. *(E.)* Impiciatore, M.; Debas, H.; Walsh, J. H.; Grossman, M. I.; Bertaccini, G. (VA Wadsworth Hosp. Cent., Los Angeles, Calif.). *Rend. Gastroenterol.* 6(2):99-101, 1974.

The effect of bombesin on serum gastrin and on acid secretion in dogs was studied to confirm previous findings. Three dogs (20-25 kg) were prepared with vagally denervated antral pouches, gastric fistulas, and Heidenhain pouches; 3 other dogs (19-25 kg) were antrectomized, and prepared with gastric fistulas, and Heidenhain pouches. Acid concentration was measured by titrating 0.2 ml samples with 2 M NaOH to pH 7.0; serum gastrin concentrations measured by radioimmunoassay. An i.v. infusion of bombesin (0.5 µg/kg/hr) for 90 min, with the antral pouch irrigated with 0.1 M $NaHCO_3$, increased serum gastrin and acid secretion in gastric fistula (107% pentagastrin response) and Heidenhain pouch (84% pentagastrin response). The responses peaked near the end of the infusion. Release of gastrin was inhibited but not abolished by bathing the antral pouch with 0.1 M HCl or by pretreating with atropine sulfate (0.2 mg/kg, s.c.). In antrectomized dogs, bombesin (1 µg/kg/hr) caused less than 1/10 gastrin release and acid secretion as in dogs with antrum. The acid response to bombesin was compared to the maximal response to pentagastrin and found similar. It is concluded that the mechanism by which bombesin stimulates acid secretion is by release of gastrin, mainly from the antrum, confirming previous studies.

1707 GASTRIN RELEASE BY BOMBESIN IN THE ANTRAL POUCH DOG. *(E.)* Basso, N.; Improta, G.; Melchiorri, P.; Sopranzi, N. (Dep. Surg. Pathol. III, Univ. Rome, Italy). *Rend. Gastroenterol.* 6(2):95-98, 1974.

Gastrin release by bombesin was studied in dogs provided with Heidenhain pouches, gastric fistulas, and innervated and denervated antral pouches. Each experiment was performed 4 times on each of 4 dogs, and blood samples (5 ml) were taken at 15 min intervals following infusion for determination of plasma gastrin. A control i.v. infusion of 0.15 M NaCl (60 ml/hr for 150 min) in dogs with an innervated antral pouch at pH 7.0 produced a consistent basal acid secretion in the Heidenhain pouch (60 µEq/15 min) and main stomach (1,000 µEq/15 min) and basal levels of immunoreactive plasma gastrin of 90 pg/ml. Acidifying the antral pouch (pH 1.0) lowered acid secretion (30 µEq/15 min) and plasma gastrin (39 pg/ml) in the Heidenhain pouch, but did not affect the stomach. Following antral denervation no acid secretion was observed in basal conditions in the stomach or Heidenhain pouch. Basal plasma gastrin was significantly lower (34 pg/ml). An insulin injection (1.6 U/kg) in dogs with innervated antral pouch produced a rise in stomach acid to a peak of 4,200 µEq/15 min. Plasma gastrin peaked at 130 pg/ml. After antral denervation insulin injection produced acid secretion from stomach, but no change in plasma gastrin. Bombesin infusion (1 µg/kg/hr) in dogs with innervated neutral antral pouch increased acid in stomach (3,800 µEq/15 min) and Heidenhain pouch (350 µEq/15 min); gastrin peaked at 280 pg/ml. Acidifying the antral pouch produced a significant but brief decrease in acid secretion and plasma gastrin followed by recovery. In dogs with denervated antral pouch, bombesin produced peak acid in stomach (2,600 µEq/15 min) and Heidenhain pouch (275 µEq/15 min) and peak gastrin of 250 pg/ml. The effect of acidifying the antral pouch was similar to that found in the innervated antral pouch. The authors conclude that gastrin is released from dog antrum following administration of bombesin, independent of vagal innervation, and bombesin appears to be the only stimulant to produce similar gastrin release from both innervated and denervated antral pouch.

1708 EFFECT OF SUPRAMAXIMAL DOSES OF STIMULI ON DEOXYRIBONUCLEIC ACID CONTENT OF GASTRIC ASPIRATE. *(E.)* Desai, H. G.; Venugopalan, K.; Antia, F. P. (B. Y. L. Nair Charitable Hosp., Bombay, India). *Indian J. Med. Res.* 62(9):1354-1359, 1974.

The DNA content of gastric juice was measured after maximal and supramaximal stimulation by pentagastrin and histamine. Normal control subjects, and subjects with duodenal ulcer and chronic gastritis had their stomach contents aspirated for 1 hr after a saline rinse, then 500 ml normal saline was injected into the stomach and aspirated. After basal values were thus determined, the process was repeated after s.c. injection of pentagastrin (300 or 450 µg) or histamine acid phosphate (2.4 or 3.2 mg). The antihistamine anthisan (50 mg, i.m.) was injected 1/2 hr before histamine injection. No significant increase in DNA was seen in 24 subjects after the lower dose of histamine, but the higher dose caused a significant increase of 5.5 ng atoms DNA-phosphorus (P)/min in 19. In 15 of the 19, a rise of DNA-P was observed. Of 12 subjects, the lower pentagastrin dose caused a barely significant increase of 3.6 ng atoms DNA-P/min. In 9 DNA-P was increased. The higher dose caused a significant rise of 4.8 in 14 subjects, in 11 of whom the DNA-P was increased. It is concluded that supramaximal doses of both pentagastrin and histamine significantly increase DNA content of gastric aspirate, indicating increased exfoliation of gastric surface epidermal cells.

1709 EFFECT OF p-CHLOROMERCURIBENZENE SULFONATE ON GASTRIC PARIETAL AND SURFACE CELL FUNCTION IN THE DOG. *(E.)* Moody, F. G.; Simons, M.; Jackson, T. (Univ. Utah Med. Cent., Salt Lake City). *Gastroenterology* 68(2):279-284, 1975.

Net ion and water movement across canine gastric secretory mucosa after exposure to *p*-chloromercuribenzene sulfonate, a sulfhydryl reagent which inhibits gastric acid secretion, was studied in mongrel dogs weighing about 15-20 kg. The dogs were anesthetized, and a segment of greater curvature of the stomach was excised and placed in a double lumen Lucite chamber. This provided 2 equal-sized mucosal compartments each with its own blood supply. Instillates were delivered into the hemichambers, recovered, analyzed for changes in volume, hydrogen, sodium and potassium, chloride, and protein. The 2 mucosal surfaces were used to compare ion and water fluxes in the presence and absence of *p*-chloromercuribenzene sulfonate (1-10 mM in mannitol). The reagent had a profound inhibitory effect on acid output after the gastric fundus had been stimulated by histamine phosphate, and this was accompanied by a large efflux of Na^+. When the reagent was applied

on Tris-buffered mannitol there was less depression of H^+ output, while sodium efflux remained large, suggesting that back-diffusion had not been prevented by the buffer. There was no change in appearance during exposure of the resting mucosa to the reagent in mannitol with or without the buffer, but there was a gradual accumulation of mucus when the reagent was instilled in isosmotic acid. There was a small increase in protein output. *p*-Chloromercuribenzene sulfonate inhibited acid secretion in response to histamine by an effect on both parietal and surface cell function, resulting in both a decrease in H^+ production and an increase in hydrogen back-diffusion.

1710 APPEARANCE OF GASTRIN IN PERFUSATES FROM THE ISOLATED GASTRIC ANTRUM OF DOGS. *(E.)* Andersson, S.; Nilsson, G. (Dep. Pharmacol., Karolinska Inst., Stockholm, Sweden). *Scand. J. Gastroenterol.* 9(7):619-621, 1974.

Three mongrel dogs were provided with Heidenhain pouches and fully innervated antral pouches in a study to determine whether the material with gastrin-like immunoreactivity in gastric juice is gastrin derived from the antrum mucosa and, if so, whether antral acidity might affect the outflow of antral gastrin into the gastric lumen. Gastric secretion was stimulated by perfusing the antral pouches with 15% ethanol solutions (100 ml/hr for 4 hr) at pH 7 or pH 1 or by a test meal consisting of 500 g of canned dog food with simultaneous perfusion of the antral pouches with buffer solutions at pH 7 or pH 1 (100 ml/hr for 3 hr). The ability of the antral perfusates to induce acid secretion was tested on Heidenhain pouch dogs during background stimulation with urecholine (0.5 mg/hr, i.v.). The presence of gastrin-like immunoreactivity in the antral perfusates was determined by radioimmunoassay. I.v. administration of extracts from the acid, but not from the neutral perfusates, evoked secretion from the gastric pouches. Gastrin-like immunoreactivity was present in both neutral and acid antral perfusates, but the concentrations and total amounts of gastrin-like activity were approximately 7 times higher in acid perfusates. The identical immunochemical behavior of the gastrin-like material and the pure porcine standard gastrin used in the immunoassay indicates that the immunoreactive material is gastrin, or is closely related to it. The relatively high concentrations of this material in perfusates from antral pouches lead to the conclusion that the major source of gastrin-like material in gastric juice may be the antrum mucosa. These results indicate that there is a "leakage" of gastrin-like activity into the antral lumen that is greatly increased at low pH's. It is suggested that this mechanism may be responsible for acid inhibition of gastrin release.

1711 RESISTANCE OF THE ANTRAL MUCOSA TO THE TROPHIC EFFECT OF GASTRIN IN THE DOG. *(E.)* Willems, G.; Vansteenkiste, Y. (St. Peter's Hosp., Brussels, Belgium). *Biol. Gastroenterol. (Paris)* 7(3):237-240, 1974.

Purified porcine gastrin was administered i.v. to adult beagle dogs (13-17 kg) to study its trophic effect on antral mucosa. A nylon gastric cannula was inserted into each of 4 dogs to allow for serial biopsies to be taken of fundic and antral mucosa. Gastrin (4 µg/kg/hr) was infused for 4 hr and secretion collected every 15 min for volume and pH determination. Mucosal specimens (60 mm^2) taken at 4, 8 and 12 hr one day, and 16, 20 and 24 hr another day following a similar infusion, were prepared for histologic examination, then covered with autoradiographic emulsion. In the fundic glands, the labeling index (% of cells in DNA synthesis) increased to 250% of its control value by the 12th hr. The mitotic index increased from the 16th hr. There was no variation in controls. Antral specimens of the same animals showed an insignificant decrease in labeling and mitotic indices and no variations in controls. It is concluded that gastrin, at a dose which elicits all its known physiologic properties, has no trophic effect on the antral mucosa.

1712 RESPONSE OF THE ISOLATED PERFUSED STOMACH OF THE DOG TO ELECTRICAL VAGAL STIMULATION. *(E.)* Lanciault, G.; Shaw, J. E.; Urquhart, J.; Adair, L. S.; Brooks, F. P. (Univ. Pennsylvania Sch. Med., Philadelphia). *Gastroenterology* 68(2):294-300, 1975.

The effect of vagal stimulation on immunoreactive gastrin release, gastric blood flow, and gastric acid output was studied in the isolated dog stomach. The isolated stomachs were perfused with the blood of an intact anesthetized dog at an input pressure of 100 mm Hg. The mucosal surface was perfused through an esophageal cannula with lactated Ringer's solution at 2 ml/min. Gastric contents were analyzed for acid secretion by titration, and arterial and venous blood were sampled regularly to determine gastrin concentrations. The left vagal trunk was stimulated with 7 V at 5 msec with the frequency varying from 0.5 to 10 Hz. Vagal stimulation at 5 Hz for 30 min was found to increase immunoreactive gastrin release and gastric blood flow significantly over control values by 88 pg/ml and 25 ml/min, resp. At 0.5, 2, or 10 Hz, immunoreactive gastrin concentrations were significantly elevated over controls, the greatest increase (4-fold) at 2 Hz. Gastric blood flow increased significantly only at 10 Hz, where it measured 70 ml/min. Gastric acid output was significantly increased at 0.5, 2 or 10 Hz (approximately 3.0-5.5 mEq/ml) over control values (which were negligible). It is concluded that isolated canine stomach responds to vagal stimulation with increase in gastric acid output, blood flow, and immunoreactive gastrin release.

1713 GASTRIC SECRETION AS A FUNCTION OF ACUTE AND CHRONIC STRESS IN THE GASTRIC FISTULA RAT. *(E.)* Pare, W. P.; Isom, K. E. (VA Hosp., Perry Point, Md.). *J. Comp. Physiol. Psychol.* 88(1):431-435, 1975.

The effects of stress on gastric acid secretion were studied using gastric cannulas. Long-Evans rats (30) were cannulated and divided into 3 groups. The

1st group received grid electric shocks after a warning stimulus, and the 2nd without warning. Both received 6 shocks/hr randomly spaced. The 3rd group was a control and received no shock. Gastric samples were taken every 2 hr for 12 hr during the 1st day, as acute stress samples. The shock treatments were continued for 6 days, and on the last day, chronic stress samples were taken in the same manner. In a 2nd experiment, with 24 rats, baseline secretion samples were taken during a 10-day pre-stress period, each rat serving as its own control. In the 1st experiment, the rats given signalled shocks secreted significantly more gastric juice during chronic samples. In the 2nd experiment, gastric secretion was depressed for acute stress in the signaled shock group and total acid output for both groups (acute). Chronic stress caused an increase in both, but only the elevation of the total acid output from the unsignalled shock group was statistically significant. It is concluded that acute stress temporarily inhibits gastric secretion, but over a period of time the rats adapt to the stress and the values return to normal.

1714 RELEASE OF GASTRIC INHIBITOR FROM THE INTESTINE OF DOGS INFUSED INTRAVENOUSLY WITH SODIUM OLEATE. *(E.)* Kowalewski, K.; Secord, D. C. (Surg. Med. Res. Inst., Univ. Alberta, Edmonton, Canada). *Pharmacology* 12(3):177-185, 1974.

The ability of extracts of intestinal secretions or mucosa from sodium oleate-infused dogs to inhibit gastric secretion was studied in Labrador Retrievers with Heidenhain pouches. Dialyzed and lyophilized intestinal secretions or mucosal extracts collected from sodium oleate (0.02M, 10ml/hr) infused dogs over a 6 hr period were dissolved in glycine buffered 2 M urea (pH 9.5) and infused i.v. simultaneously with pentagastrin (6 µg/kg/hr) for 6 hr (20 mg/hr or 60 mg/hr). Extracts from saline-infused dogs served as controls. Intestinal secretion and mucosal extracts from oleate infused dogs inhibited gastric secretion by 62% and 47%, resp. Secretory extracts produced a 12% ($P < 0.01$) decrease in gastric H^+ concentration whereas mucosal extracts had no effect on this parameter. Infusion of sodium oleate had no effect on the composition of blood nor did it produce any changes in bile and pancreatic juice. Except for a slight decrease in liver cholesterol ($P < 0.05$), hepatic and pancreatic fatty acid composition was also not altered by oleate infusion. None of the dogs developed a febrile response, hypotension or vomiting during infusion of the extract, suggesting that the inhibition of gastric secretion in these animals was a specific effect. It is concluded that an enterogastrone-like inhibitor can be released in dogs by i.v. sodium oleate infusion as well as by intestinal fat instillation as has previously been shown.

1715 THE ROLE OF THE UPPER DUODENUM IN CONTROLLING GASTRIC ACID SECRETION IN THE RAT. *(E.)* Lundberg, B.; Andersson, S. (Dep. Pharmacol., Karolinska Inst., Stockholm, Sweden). *Scand. J. Gastroenterol.* 9(7):623-627, 1974.

The effects of 3 types of duodenal ligation on gastric secretory volume, acidity, acid output, and ulcer frequency in Sprague-Dawley rats were compared with that of pylorus ligation. The time between ligation and sacrifice varied between 3 and 16 hr. The 4 types of ligation used were: 1) ligature placed exactly at the pyloric sphincter; 2) ligature placed immediately proximal to the entrance of the common bile duct; 3) ligature around the duodenum half way between the pylorus and the bile duct; and 4) ligature placed so that the distance from the pylorus to the ligature was 1/4 of that from the pylorus to the entrance of the bile duct. There was no change in gastric juice volume 16 hr after ligation, whereas acidity and acid output were progressively reduced the further away from the pylorus the ligature was placed. Rats with duodenal ligatures had fewer and less severe ulceration compared with pylorus-ligated rats. With shorter periods of ligation (3 or 5 hr), duodenal ligation reduced both volume and acid output by about the same order of magnitude, independent of where the duodenal ligature was placed. The lowered acid output in rats with different duodenal ligations is probably the main reason for the smaller number of ulcers and perforations in comparison with pylorus-ligated groups. It is concluded that the upper duodenum of the rat contains a pH-sensitive gastric inhibitory mechanism.

1716 THE EFFECT OF A NEW BENZODIAZEPINE PSYCHOPHARMACEUTICAL AGENT, BROMAZEPAM, ON BASAL GASTRIC ACID SECRETION. *(Ger.)* Stacher, G. (1st Surg. Clin., Univ. Vienna, Austria). *Z. Gastroenterol* 12(3):179-184, 1974.

1717 EFFECT OF PROSTAGLANDIN ON GASTRIC SECRETORY FUNCTION. *(Pol.)* Swiderska-Kulikowa, B. (Acad. Med., Warsaw, Poland). *Pol. Arch. Med. Wewn.* 52(3):299-303, 1974.

1718 THE EFFECT OF SECRETAGOGUES AND ANTISECRETAGOGUES ON HISTIDINE DECARBOXYLASE ACTIVITY IN RAT GASTRIC MUCOSA. *(E.)* Oscarson, J.; Hakanson, R.; Liedberg, G. (Dep. Pharmacol., Univ. Lund, Sweden). *Agents Actions* 4(3):183-184, 1974.

1719 EFFECT OF ACIDIFICATION OF TOTALLY DENERVATED POUCHES OF THE DUODENAL BULB ON GASTRIC ACID SECRETION IN DOG. *(E.)* Nilsson, G. (Dep. Pharmacol., Karolinska Inst., Stockholm, Sweden). *Acta Physiol. Scand.* 92(4):569-571, 1974.

1720 THE INFLUENCE OF ACID AND BILE ON THE VIABILITY OF RABBIT MUCOSA. *(E.)* Fiddian-Green, R. G.; Silen, W. (no affil.) *S. Afr. J. Surg.* 12(2):69, 1974.

1721 ACID BASE BALANCE IN RELATION TO GASTRIC ACID SECRETION STIMULATED BY HISTAMINE AND PENTAGASTRIN IN CATS. *(E.)* Albinus, M.; Sewing, K. F. (Pharmacol. Inst., Univ. Tubingen, Germany). *Naunyn Schmiedebergs Arch. Pharmacol.* 285(4):325-335, 1974.

1722 THE EFFECT OF HISTAMINE-H_2-RECEPTOR-ANTAGONISTS ON GASTRIC ACID SECRETION, GASTRIC MUCOSAL BLOOD FLOW AND ACID BASE BALANCE IN CATS. *(E.)* Albinus, M.; Sewing, K. F. (Pharmacol. Inst., Univ. Tubingen, Germany). *Naunyn Schmiedebergs Arch. Pharmacol.* 285(4):337-353, 1974.

1723 EFFECT OF α- AND β-ADRENERGIC BLOCKING AGENTS ON THE DEVELOPMENT OF NEUROGENIC DYSTROPHY OF THE GASTRIC WALL AND ON GASTRIC CATECHOLAMINE CONTENT. *(Rus.)* Linich, E. P.; Zabrodin, O. N. (Inst. Exp. Med., Leningrad, USSR). *Farmakol. Toksikol.* 37:452-455, 1974.

1724 EFFECT OF ULCER-PROTECTING AGENTS ON THE FREE AMINO ACID CONTENT OF THE MUCOSA IN VARIOUS AREAS OF THE STOMACH IN EXPERIMENTAL NEUROGENIC DYSTROPHY. *(Rus.)* Mirzoian, S. A.; Tatevosian, A. T. (Erevan Med. Inst., USSR). *Zh. Eksp. Klin. Med.* 14(1):3-8, 1974.

1725 SENSING OF THE SECRETORY PROCESS BY A RECEPTOR APPARATUS IN THE GASTRIC MUCOSA. *(Rus.)* Bagaev, V. A.; Kurtsin, I. T.; Nozdrachev, A. D. (I. P. Pavlov Inst. Physiol., Leningrad, USSR). *Dokl. Akad. Nauk SSSR* 220(2):489-492, 1975.

1726 ACTION MECHANISM OF VEGETABLE JUICES ON PROPERTIES OF GASTRIC JUICE. *(Rus.)* Mukhin, I. M.; Zarembskii, R. A.; Mozzhukhin, A. S. (S. M. Kirov Mil. Med. Acad., Leningrad, USSR). *Fiziol. Zh. SSSR* 40(12):1891-1892, 1974.

1727 GASTRIN: SOME RECENT ASPECTS. *(Fr.)* de Graef, J.; Woussen-Colle, M. C.; Limbosch, J. M. (St. Pierre Univ. Hosp., Brussels, Belgium). *Acta Gastroenterol. Belg.* 37(6):247-258, 1974.

1728 OPT-REACTIVE CELLS AND GASTRIN CELLS IN THE DIGESTIVE TRACT OF THE CHICKEN. *(E.)* Sundler, F.; Larsson, L. I.; Hakanson, R.; Rehfeld, J.; Holst, J.; Stadil, F. (Dep. Histol., Univ. Lund, Sweden). *Agents Actions* 4(3):208-209, 1974.

1729 EXPERIMENTAL HYPERGASTRINAEMIA AND ANTRAL-GASTRIN-CELL HYPERPLASIA IN DOGS. *(E.)* Jennewein, H. M.; Ganguli, P. C.; Waldeck, F.; Siewert, R.; Polak, J. M.; Pearse, A. G. E. (Biol. Pharm. Res., C. H. Boehringer Sohn, Ingelheim/Rhine, Germany). *Gut* 15(10):835-836, 1974.

1730 IN VITRO BIOSYNTHESIS OF GASTRIC MUCOSAL MACROMOLECULES. *(Fr.)* Waldron-Edward, D.; Decaens, C.; Bader, J. P.; Robert, L. (Henri Mondor Hosp., Creteil, France). *C. R. Acad. Sci. [D] (Paris)* 279:1397-1399, 1974.

1731 PROTEIN SYNTHESIS AND SECRETION IN CULTURED RABBIT GASTRIC MUCOSAL BIOPSIES. *(E.)* Sutton, D. R.; Donaldson, R. M. (Nottingham Gen. Hosp., England). *Gut* 15(10):836, 1974.

1732 CONDIMENTS. *(E.)* Demling, L.; Koch, H. (Med. Clin., Univ. Erlangen-Nuremberg, Erlangen, Germany). *Acta Hepatogastroenterol. (Stuttg.)* 21(5):377-379, 1974.

See also, 1754, 2005, 2007.

SECRETION AND METABOLISM

Pancreas

1733 THE EFFECTS OF CHOLECYSTOKININ-PANCREOZYMIN, ACETYLCHOLINE AND SECRETIN ON THE MEMBRANE POTENTIALS OF MOUSE PANCREATIC CELLS IN VITRO. *(E.)* Greenwell, J. R. (Med. Sch., Univ. Newcastle upon Tyne, England). *Pfluegers Arch.* 353(2):159-170, 1975.

Cell membrane potentials in an *in vitro* preparation of pancreas from CBA mice of either sex were measured and the effects of cholecystokinin-pancreoxymin, acetylcholine, and secretin studied. The pancreas was placed in a tissue bath through which there was a flow of warmed superfusion fluid; the connective tissue sheath on the upper surface of the pancreas was removed by microdissection. The excised pancreas was allowed to equilibrate in the superfusion fluid for 20 min and the potentials recorded throughout the succeeding 3 hr. A microelectrode was advanced for distances up to 200 μ into the pancreatic tissue in areas over the duct system. The calibration pulse height and membrane potential deflection were fed into a computer which calculated the microelectrode resistance and the cell membrane potential, and printed out the membrane potential-frequency histogram. This histogram had 4 distinct peaks and was split into the sum of 4 Gaussian distributions with means of -14, -23, -32, and -41 (mV). The -32 mV peak was attributed to acinar cells which respond to cholecystokinin-pancreozymin (10^{-2} to 10^{-1} U/ml) or acetylocholine (10^{-7} to 10^{-5} M) by depolarizing to -15 to -20 mV, the response lasting up to 10 min. The -23 and -41 peaks were due to duct cells, which had a resting membrane potential of -23 mV and hyperpolarized to -41 mV in response to secretin stimulation. This response lasted for more than 15 min.

Duct cells, regardless of their position in the duct system, functioned electrophysiologically in identical fashion. It is concluded that the secretory sites for enzyme and electrolyte are distinct, the duct cells secreting only electrolyte and the acinar cells only enzyme.

1734 THE EFFECT OF AN INTRADUODENAL ELEMENTAL DIET ON PANCREATIC SECRETION. *(E.)* Wolfe, B. M.; Keltner, R. M.; Kaminski, D. L. (Dep. Surg., St. Louis Univ., Mo.). *Surg. Gynecol. Obstet.* 140(2): 241-245, 1975.

The effect of an elemental diet on canine exocrine pancreatic secretion was evaluated by infusing an elemental diet into the duodenum and monitoring the output of the pancreas. Four dogs were prepared with pancreatic and chronic gastric fistulas. Perfusates used in these experiments were 0.15 M NaCl with an osmolality of 285 mOsm/liter, pH 6.9; 0-16 normal HCL in 0.15 M NaCl, osmolality of 290 mOsm/liter, pH 1.6; 127 mM L-phenylalanine with an osmolality of 300 mOsm/liter, pH 7.0. The elemental diet perfusate was prepared by diluting 80 g commercial vivonex 100 in 300 ml water to give a solution with a pH of 5.4 and a mean osmolality of 890 mOsm. The calculated amino acid concentration of the elemental diet infused was 155 mM/liter and that of the L-phenylalanine solution was 127 mM/liter, resulting in an amino load of 5.8 mOsm/15 min when the elemental diet was infused and 4.5 mOsm/15 min when the L-phenylalanine solution was infused. Cholecystokinin, when appropriate, was administered by continuous i.v. infusion, 2 U/kg/hr. Pancreatic secretions were collected and measured every 15 min. Pancreatic protein concentration was equated with pancreatic enzyme concentration and was determined by measuring fluorescence at 280 nm. Intraduodenal L-phenylalanine and elemental diet produced significant increases in pancreatic output and protein concentration when compared with control values. Both elemental diet and L-phenylalanine perfusion produced a pancreatic volume response not significantly different from that produced by cholecystokinin. The concentration of bicarbonate was significantly altered from control values by intraduodenal infusion of phenylalanine and elemental diet and i.v. administration of cholecystokinin. These findings suggest that the stimulatory properties of the elemental diet are primarily due to the amino acid content and that it stimulates the duodenal mucosa to secrete cholecystokinin which stimulates the exocrine pancreas. It is concluded that elemental diets administered in such a manner that the substance passes through the duodenum would not be a satisfactory method of providing calories to patients with acute pancreatitis.

1735 PANCREATIC ATROPHY AND OTHER PATHOLOGICAL CHANGES IN RATS FOLLOWING THE FEEDING OF STANNOUS CHLORIDE. *(E.)* Dreef-Van der Meulen, H. C.; Feron, V. J.; Til, H. P. (Cent. Inst. Nutr. Food Res., Zeist, Netherlands). *Pathol. Eur.* 9(3):185-192, 1974.

The pathological effects of tin in rats were studied by feeding stannous chloride in levels just under those needed to produce growth retardation. Male and female weanling Wistar rats were maintained for 13 weeks on basal diets or basal diets containing stannous chloride; the dietary level of the tin compound was increased for 0.1-0.8% during the first 8 weeks, and held constant at 0.8% thereafter. None of the animals died during the experiment and none showed abnormal behavior. After the 4th week, the tin-fed males showed a reduction in weight gain and a lower food intake compared with the controls; no such changes were noted in the females. Hemoglobin levels in the blood were significantly decreased from week 6 in the males and from week 10 in the females. Mean cell volume, mean cell hemoglobin, and mean cell hemoglobin concentration were decreased in all tin-fed animals, the effect being greatest in the males. Alkaline phosphatase activities were decreased significantly in the females and nonsignificantly in the males. The relative weights of the kidneys, liver and adrenals were increased in both sexes and the weights of the heart, brain, testicles, and thyroid were increased in the males. The pancreas was often reduced in size and showed a wide spectrum of degenerative changes. The gastrointestinal tract in general showed gross evidence of irritation, and the small intestine underwent hypertrophic and hyperplastic changes in all tin-fed animals. Striking hepatic pallor occurred in 1 male, and enlarged hepatocytes and histological changes in the bile duct epithelium were noted in some males. Pancreatic atrophy appears to be the most specific morphological manifestation of the toxic action of stannous chloride in the doses given.

1736 PANCREATIC ENZYME SECRETION IN THE CONSCIOUS RAT. METHOD AND APPLICATION. *(E.)* Hotz, J.; Zwicker, M.; Minne, H.; Ziegler, R. (Cent. Intern. Med., Univ. Ulm, Germany). *Pfleugers Arch.* 353(2):171-189, 1975.

A method developed for collecting duodenal juice and gastric content separately in conscious rats was used in secretory studies in the resting state and in response to cholinergic and hormonal stimuli, as well as under the influence of narcosis. Stainless steel cannulas were implanted into the stomach fundus of male albino Wistar rats and a double lumen tube inserted through the cannula *via* the pylorus into the duodenum. The 2 external openings of the gastric and duodenal tubes were connected to 2 collection vials. In the resting state (11 animals) there was a constant flow rate of duodenal volume, bicarbonate, trypsin, and amylase. Cholinergic stimuli (8 animals; carbachylcholine, 0.01 mg/100 g body weight) were capable of increasing enzyme secretion as much as 4-fold for 30-40 min when administered as a single s.c. injection. This effect was annulled by atropine (8 rats; 0.001 mg/100 g body weight). Secretin and cholecystokinin-pancreozymin (2.5 U/100 g body weight, each hormone) given together in a single injection s.c. or i.v. cause a similarly strong response (10 animals). The responses increased from 0.35 ml/20 min to a peak rate of 0.60 (s.c.) and from 0.42 to 0.65 (i.v.), resp. Identical ranges of the secretion maxima were found, with a tendency to decrease after the lst hr, when the hormones were infused either s.c. or i.v. (49 animals). Doses from 0.5 to 25 U/100 g body weight/hr showed identical responses.

Doses below 0.2 U/100 g/hr were without effect. In 9 animals narcosis was initiated after a resting period of 1 hr by a single i.m. injection of 4 mg/100 g body weight phenobarbital. A marked depression of the resting secretion of volume and enzyme concentration by about 50% resulted. Secretin and cholecystokinin-pancreozymin, given by i.v. infusion (2.5 U/100 g body weight/hr, each hormone) caused a peak enzyme secretion rate which was significantly below the values of the animals tested in the conscious state. It is concluded that the method of the separate duodenal aspiration of the conscious rat seems to be suitable for examination of physiologic and pharmacologic effects on the resting and on the stimulated pancreatic enzyme secretion. Several tests can be performed on the same animal over a more than 3-week period, permitting the study of acute as well as chronic effects in the same animal.

1737 EFFECT OF EXPERIMENTAL HYPERTHYROIDISM ON PHOSPHOLIPASE A_2, LIPASE, CHOLINESTERASE, AND TRYPSIN ACTIVITIES IN THE PANCREATIC JUICE AND PANCREAS OF THE RAT. *(Fr.)* Belleville, J. (Fac. Sci. Mirande, Dijon, France). *J. Physiol. (Paris)* 68(1):65-74, 1974.

Hyperthyroidism was produced in Wistar rats by daily i.p. injection of thyroxine (50 µg/100 g) for 2 weeks. Pancreatic juices were obtained from 12 control and 9 hyperthyroid rats, and the pancreas was removed and analyzed from 14 control and 12 hyperthyroid rats. Protein content of the pancreatic juice and extract was determined by the Lowry colorimetric method, DNA content by the Dische method. Thyroxine injections produced decreased body weight and increased O_2 consumption. The hyperthyroid rats showed increased total pancreatic juice output, and the pancreatic juice had a higher specific activity of phospholipase A_2 and of trypsin, and higher content of these 2 enzymes and of cholesterolesterase; they also had a lower specific activity of cholesterolesterase and of lipase and lower lipase content. Hyperthyroidism produced greater weight of the pancreas, higher ratio of the pancreas to body weight, and, in the pancreas extract, increased total amount of protein, increased total amount of the 4 enzymes, increased total amount of DNA, significant increase in the specific activity relative to protein content for all enzymes except cholesterolesterase, and increased specific activity relative to DNA content for all 4 enzymes. It was concluded that thyroxine injections cause increased protein synthesis by the pancreas. The parallel increase in weight of the pancreas and amount of DNA suggests that there is an increase in the number of cells in the pancreas. It appears, judging from the case of lipase, that the excretion of pancreatic enzymes is not always proportional to the amount of enzyme in the pancreas.

1738 IN VITRO INTERACTIONS OF GASTROINTESTINAL HORMONES ON CYCLIC ADENOSINE 3'5'-MONOPHOSPHATE LEVELS AND AMYLASE OUTPUT IN THE RAT PANCREAS. *(E.)* Deschodt-Lanckman, M.; Robberecht, P.; De Neef, P.; Labrie, F.; Christophe, J. (Univ. Brussels Med. Sch., Belgium). *Gastroenterology* 68(2): 318-325, 1975.

The effects of secretin, vasoactive intestinal polypeptide, pancreozymin (PZ), cerulein, C-terminal octapeptide of PZ (OC-PZ), and glucagon, singly and in combination, on cyclic AMP and amylase levels (per mg protein) in pancreas fragments from adult male Wistar rats are presented. Theophylline (10 mM) was added to the incubating solutions to amplify the cyclic AMP response. The 3-, 4-, and 5-fold increases in cyclic AMP levels observed 10 min after incubation with 10^{-7} M secretin, 10^{-6} M vasoactive intestinal polypeptide and 10^{-7} M secretin and 10^{-6} M vasoactive intestinal polypeptide, resp., were inhibited by 10^{-7} M PZ, cerulein, or OC-PZ (noncompetitive inhibition was suggested by the data but not proven). Significant amylase output, potentiated by secretin but not vasoactive intestinal polypeptide, was observed with 10^{-9} M PZ, cerulein, and OC-PZ. There was no correlation between amylase output and cyclic AMP levels. Copious amylase secretion was elicited even at control levels of cyclic AMP. Glucagon exerted no effect on either system. The data indicate that if an excess of cyclic AMP is necessary for amylase hypersecretion from acinar cells under physiological concentrations of pancreozymin, the action of this hormone must be limited to a small pool of cyclic AMP with a high turnover.

1739 EXOGENOUS AND ENDOGENOUS SECRETIN STIMULATION IN THE CONSCIOUS RAT. *(E.)* de Smul, A.; de Waele, B.; Wissocq, P.; Kiekens, R. (Lab. Exp. Surg., Vrije Univ., Brussels, Belgium). *Digestion* 11(1/2):39-50, 1974.

The effects of exogenous and endogenous secretin stimulation on the characteristics of pancreatic secretion were studied in male Wistar rats maintained in normal states of hydration and internal temperature. In the exogenous secretin experiments, secretin (1-8 U/kg) was injected i.v. at 3-hr intervals and the pancreatic juice collected in successive 10-min periods. In the endogenous secretin experiments, 0.1 N HCl or isotonic NaCl was infused in the duodenum at rates of 1-16 ml/hr, and samples of pancreatic juice were collected every 30 min. In the exogenous experiments, the mean basal secretion of pancreatic juice (8.85 µl/min) was significantly increased by secretin administration; higher doses of secretin prolonged the stimulatory effect. The mean basal bicarbonate concentration (20.0 mEq/liter) was moderately increased (compared with other species) by secretin administration, while the mean bicarbonate output (0.175 µEq/min) was increased even by low doses of secretin; higher doses prolonged the stimulatory effect. Secretin in high doses caused a drop in the protein concentration 10-20 min after administration; a significant increase in protein output occurred during the first 10 min after secretin injection. In the endogenous experiments, the volume of the pancreatic secretion rose moderately but significantly during infusion of 0.4, 0.8 and 1.6 mEq HCl/hr. The mean basal bicarbonate concentration increased slightly after HCl infusion, while the mean basal bicarbonate output rose significantly. The protein concentration decreased with HCl infusion and the protein output did not

change. NaCl infusion caused no significant changes in volume, bicarbonate, or protein. The maximal secretory response to endogenous stimulation was less than after i.v. secretin injection, the rate of secretin release being the limiting factor in the endogenously stimulated secretin. It is concluded that postoperative basal pancreatic secretion is high in conscious rats, even when endogenous secretin stimulation induced by intraduodenal emptying of gastric acid is prevented by pylorus ligation. It is further concluded that the maximal response to secretin stimulation in rats is low in comparison with other species.

1740 STUDIES ON THE ELIMINATION OF SECRETIN AND CHOLECYSTOKININ WITH REGARD TO THE KINETICS OF EXOCRINE PANCREATIC SECRETION. *(E.)* Lehnert, P.; Stahlheber, H.; Forell, M. M.; Fullner, R.; Fruhauf, S.; Fritz, H.; Hutzel, M.; Werle, E. (2nd Med. Hosp., Univ. Munich, Austria). *Digestion* 11(1/2):51-63, 1974.

The extent of secretin and cholecytsokinin inactivation by the liver and kidneys and the kinetics of pancreatic secretion were studied in 72 fasted mongrel dogs. Under pentobarbital anesthesia, the pancreatic duct was drained ahead of the point where it opens into the duodenum, accessory pancreatic ducts were ligated, the common bile duct was drained, the pylorus was ligated, venesection was performed on both femoral veins, the renal artery was exposed, and polyethylene tubes were inserted into the portal vein and a pancreatic branch of the superior pancreatocoduodenal artery. On injection or infusion of secretin into the portal vein, the pancreatic volume and bicarbonate response were significantly smaller than following secretin administration through the femoral vein. But only when secretin was administered in doses of 0.25 U/kg/hr or less. No difference was observed in the secretion of pancreatic protein or enzyme after administration of cholecystokinin *via* the portal or femoral vein. Compared with femoral administration, biological activity in the pancreatic juice was reduced by 75 and 60% following administration of secretin and cholecystokinin, resp., through the renal artery. Ligature of the blood vessels of both kidneys resulted in an 80% increase in the biological activity of secretin and a 50% increase in the activity of cholecystokinin. The slopes of the flow rate curves after secretin injection were smaller after ligature of the renal blood vessels, and the biological half-life of secretin increased from 2.9-4.8 min. The data indicate that the kidneys play a role in the catabolism of exogenous secretin and cholecystokinin, while the blood and liver seem to play little part, at least with regard to cholecystokinin catabolism.

1741 INFLUENCE OF ORAL CONTRACEPTIVES ON THE RAT EXOCRINE PANCREAS: A HISTOLOGIC, HISTOCHEMICAL, AND ELECTRON MICROSCOPIC STUDY. *(E.)* Nizze, H. (Inst. Gen. Pathol., Univ. Rostock, Germany). *Exp. Pathol.* 9(5/6):311-316, 1974.

The influence of oral contraceptives (Ovosiston, Non-Ovlon, Sequenz-Ovosiston) on the secretion kinetics of the exocrine pancreas of rats was studied histologically, histochemically, and electron microscopically. Seventy-six Wistar rats of both sexes were divided into 4 groups: 20 given Ovosiston for 1, 3, 7, 14, 21 days; 20 given Non-Ovlon for same number of days; 24 given Sequenz-Ovosiston for 1, 3, 7, 14, 17, 19, 21 days; 12 given 0.2 g lactose for the same time periods and serving as controls. Each pill of Ovosiston contained 2.0 mg chlormadinon acetate for gestagen and 0.08 mg mestranol for estrogen; each pill of Non-Ovlon contained 1.0 mg norethisteron acetate for gestagen and 0.06 mg ethynylestradiol for estrogen; Sequenz-Ovosiston was simply 0.10 mg mestranol when given the first 14 days and then simply Ovosiston when given the next 7 days. Each rat received a single pill dissolved in physiological saline and administered by means of a throat probe. The dose averaged 400 times that given daily to an average woman for hormonal contraception, and was found to cause no damage in rat exocrine pancreas in experiments up to 21 days. Secretory activity of acinar cells decreased in female rats early in the experiments with Ovosiston and Non-Ovlon; it increased late in the tests with Ovosiston and Sequenz-Ovosiston. Ultrastructural changes (vesiculation of endoplasmic reticulum) following Sequenz-Ovosiston could not be correlated with histochemical findings which were heterogeneous. The alterations observed are interpreted as an adaptive reaction of the secretion mechanism of the exocrine pancreas to the hormonal contraceptives, and the author suggests further studies to determine whether those changes can turn to dystrophia.

1742 INHIBITION OF EXTERNAL PANCREATIC SECRETION BY EXTRACTS OF ILEAL AND COLONIC MUCOSA. *(E.)* Harper, A. A.; Hood, A. J. C.; Mushens, J.; Smy, J. R. (Sch. Pharm., Sunderland Polytech., England). *Gut* 15(10):825, 1974.

1743 INHIBITION OF PANCREATIC SECRETION BY CALCITONIN. *(E.)* Appert, H. E.; Nakashima, Y.; Howard, J. M. (Med. Coll. Ohio, Toledo). *Fed. Proc.* 34(3):454, 1975.

1744 INHIBITION OF PANCREATIC AMYLASE RELEASE IN-VITRO BY VINBLASTINE. *(E.)* Williams, J. A. (Dep. Physiol., Univ. California, San Francisco). *Fed. Proc.* 34(3):454, 1975.

1745 INCREASE IN PANCREATIC RNA SYNTHESIS FOLLOWING STIMULATION. *(E.)* Rajotte, D.; Morisset, J. (Gastrointest. Res. Unit, Fac. Sci., Sherbrooke Univ., Quebec, Canada). *Fed. Proc.* 34(3):454, 1975.

1746 NEUROHORMONAL CONTROL OF PANCREATIC SECRETION. *(E.)* Konturek, S. J. (Med. Acad. Krakow, Poland). *Acta Gastroenterol. Belg.* 37(6):273-282, 1974.

1747 THE EFFECT OF ACUTE ALCOHOL ADMINISTRATION ON EXOCRINE PANCREATIC SECRETION IN THE PIG. *(E.)* Wheatley, I. C.; Terblanche, J.; Hickman, R.; Barbezat, G. O. (Dep. Surg., Univ. Cape Town, South Africa). *S. Afr. J. Surg.* 12(2):71, 1974.

1748 PANCREATIC CHANGES INDUCED BY CHRONIC (TWO YEARS) ETHANOL TREATMENT IN THE DOG. *(E.)* Tiscornia, O. M.; Palasciano, G.; Sarles, H. (Dig. Pathol. Res. Unit, INSERM, Marseille, France). *Gut* 15(10):839, 1974.

1749 RESPONSE OF THE RAT PANCREAS TO INTESTINAL INFUSIONS OF PHENYLALANINE, TRYPTOPHAN, LYSINE OR CASEIN. *(E.)* Lyman, R. L.; Smith, L.; Chang, I. (Dep. Nutr. Sci., Univ. California, Berkeley). *Fed. Proc.* 34(3):917, 1975.

1750 EFFECT OF BILE OBSTRUCTION IN RATS ON PANCREATIC ENZYME SECRETION AND ON RECOVERY OF TRYPSIN AND CHYMOTRYPSIN FROM THE INTESTINE. *(E.)* Green, G. M.; Nasset, E. S. (Child. Hosp. Med. Cent., Oakland, Calif.). *Fed. Proc.* 34(3):394, 1975.

1751 HISTONE MODIFICATIONS IN THE RAT PANCREAS AFTER PARTIAL PANCREATECTOMY. *(E.)* Varricchio, F.; Mabogunje, O.; Fitzgerald, P. J. (Mem. Sloan-Kettering Cancer Cent., New York, N.Y.). *Fed. Proc.* 34(3):872, 1975.

1752 COMPARISON OF ENZYME SECRETION AND MORPHOLOGY IN THE RAT PANCREAS AFTER LIGATION OF THE PANCREATIC DUCT. *(Ger.)* Schmitz-Moormann, P.; Kirchner, R. (Pathol. Inst., Univ. Marburg, Germany). *Z. Gastroenterol.* 12(1):9-14, 1974.

See also, 1692, 1851, 1871, 2150.

SECRETION AND METABOLISM

Hepatobiliary Tract

1753 HEPATIC REGENERATION IN THE ABSENCE OF PORTAL VISCERA. *(E.)* Whittemore, A. D.; Kasuya, M.; Voorhees, Jr., A. B.; Price, Jr., J. B. (Coll. Physicians Surg., Columbia Univ., New York, N.Y.). *Surgery* 77(3):419-426, 1975.

To investigate the influence of portal factors on hepatic regeneration in the rat and to clarify the apparent regulatory role of glucagon, a rat preparation was developed which was totally devoid of portal viscera and thus deficient in all possible hepatotrophic substances of portal origin. Male Sprague-Dawley rats were subjected to sham surgery (placement of catheters in the superior vena cava and the common bile duct), hepatectomy, portacaval anastomosis with hepatectomy, evisceration with hepatectomy, or evisceration with hepatectomy followed by postoperative supplementation with glucagon (0.2 mg/kg/day). The animals were killed 24-96 hr postoperatively and and representative specimens from the hepatic remnant subjected to biochemical analysis. Although the hepatic glycogen concentrations were variable, the highest levels were attained within 24 hr prior to the maximum rate of DNA synthesis in all groups except those subjected to evisceration with hepatectomy. During the following 24 hr, the glycogen level decreased concurrently with the onset of maximal DNA synthetic activity. The peak DNA synthetic response in all groups was also associated with the peak synthesis of intracellular RNA; beyond the DNA synthetic peak, RNA decreased with a gradual increase in DNA. The sham-operated rats showed a significant increase in DNA synthesis at 48 hr postoperatively (specific activity, 18 x 10^{-3} disintegrations/min/mg), DNA synthesis peaked at 36 hr in the hepatectomized rats (395 x 10^{-3}), at 72 hr in the animals subjected to portacaval anastomosis with hepatectomy (365 x 10^{-3}) and at 96 hr in those subjected to evisceration with hepatectomy (173 x 10^{-3}). The eviscerated hepatectomized rats supplemented with glucagon showed a peak in DNA synthesis at 48 hr (318 x 10^{-3}) with a larger peak at 72 (428 x 10^{-3}); these values are significantly higher than those of the eviscerated-hepatectomized rats (26 x 10^{-3} and 92 x 10^{-3} at 48 and 72 hr, resp.). The magnitude of DNA synthetic activity was similar in all groups, representing a 70-90-fold increase over baseline levels. The data suggest that glucagon facilitates and foreshortens the presynthetic phase of the hepatocellular cycle prior to the mitosis required for regeneration.

1754 CHANGES IN LIVER AND GASTRIC MUCOSAL HEXOSAMINE SYNTHESIS AFTER RESTRAINT. *(E.)* Sander, L. D.; Chandler, A. M.; Johnson, L. R. (Univ. Texas Med. Sch., Houston). *Gastroenterology* 68(2): 285-293, 1975.

The specific activity of L-glutamine:D-fructose-6-phosphate aminotransferase was measured by the method of Kornfeld in the oxyntic gland mucosas and livers of fasted (18 hr) male Stanley-Gumbreck rats after 1.5-3 hr of immobilization at 4 C. The results were compared with those obtained in rats immobilized at room temperature, in adrenalectomized rats immobilized for 1 hr at 4 C, and in rats which were injected s.c. with 5 mg/kg atropine sulfate prior to immobilization at 4 C for 3 hr. Immobilization of normal fasted rats for 3 hr resulted in a 100% frequency of lesion formation (ulceration) associated with decreased enzyme activity in the oxyntic gland

mucosa (70.1% control values) and in the liver (25.2% control). The enzyme activity returned to control levels in the stomach 9 hr after immobilization and in the liver 21 hr after immobilization. Immobilization of normal fasted rats for 1.5 hr decreased the oxyntic gland mucosa enzyme activity to 21.0% of control values, although the frequency of lesion formation was only 62.5%; the liver enzyme activity was 51.3% of control values. The concentration of UDP-N-acetylhexosamines, feedback inhibitors of this enzyme, were not altered in either tissue. Maintenance of unrestrained rats in a cold environment for 3 hr and immobilization of rats at room temperature for up to 20 hr caused neither lesion formation nor enzyme activity changes in either tissue. Adrenalectomy, which increased the damage after 1 hr of immobilization, enhanced the decreases in enzyme activity in both tissues, while atropine sulfate, which decreased the frequency of damage after 3 hr of immobilization, had no significant effect on the enzyme activity. Thus, immobilization of rats in a cold environment decreases or alters the synthesis of hexosamine-containing compounds through a mechanism not dependent on acid secretion or adrenocorticoids. This alteration in synthesis may result not only in abnormal mucus secretion, but also in altered cell membrane structure and function.

1755 NITROGEN METABOLISM IN THE PERFUSED CIRRHOTIC LIVER OF THE RAT. *(E.)* Henley, K. S.; Clancy, P. E.; Laughrey, E. G.; Lyra, L. G. C. (Univ. Michigan Med. Sch., Ann Arbor). *Lab. Clin. Med.* 85(2):273-280, 1975.

The isolated perfused liver of the cirrhotic rat was used to study the effect of ethanol and nutrition on the uptake of ammonia, or the fate of alanine, in the cirrhotic liver. Male Sprague-Dawley rats were maintained for 6 months on a cirrhogenic diet or normal laboratory diet (control). Some of the rats maintained on the cirrhogenic diet were given a normal diet for 10 days before perfusion (prefed group). Prefeeding significantly lowered the endogenous ammonia output by cirrhotic livers but did not restore the ammonia output to levels found in normal livers. During the 5-20 min perfusion period, the output of urea was also significantly lower in cirrhotic animals. Addition of ammonia to the perfusate invariably increased the output of urea. Ornithine increased both the uptake of ammonia and the output of urea in controls. In cirrhotic animals, ammonia was released into the perfusate rather than taken up by the liver, and the addition of ornithine had a nonsignificant effect in lowering the rate of ammonia release, ornithine did not increase the output of urea. Prefeeding restored the uptake of ammonia to control levels but did not restore the ability to increase the output of urea in the presence of ornithine. The addition of ethanol to the perfusate lowered the rate of uptake of ammonia in controls but did not significantly alter the output of urea. Ethanol lowered the output of urea in cirrhotic livers and nonsignificantly increased the output of ammonia. Ethanol changed neither the uptake of ammonia nor the output of urea in prefed animals. In the presence of aniline the rate of output of urea by cirrhotic and prefed cirrhotic liver was lower than that of normal livers, while the rate of ammonia output increased progressively from normal, to the prefed, to the cirrhotic liver. In cirrhotic and prefed rats, ethanol increased the amount of nitrogen recovered from alanine without significantly changing its uptake; normal livers showed an increase in the output of ammonia and glutamine. Ethanol decreased the formation of glucose by normal and prefed rats, but did not affect its formation in cirrhotic livers perfused with ammonia and ornithine. The results indicate that the cirrhotic rat liver is a source of endogenous ammonia and that the magnitude of that contribution is modified by diet and ethanol.

1756 STUDY OF THE ROLE OF THE SPLANCHNIC ORGANS IN HEPATIC ATROPHY FOR THE PURPOSE OF AUXILIARY TRANSPLANTATION OF THE LIVER. *(E.)* Serrou, B.; Michel, H.; Gelis, C.; Solassol, C.; Pujol, H.; Romieu, C. (St. Eloi Hosp., Montpellier, France). *Ann. Surg.* 180(3):274-278, 1974.

An attempt was made to identify substances in the blood which might be responsible for hepatic atrophy in auxiliary transplantation of the liver. Experimental operations were undertaken based on a variety of shunted blood vessels and the excision of isolated or associated organs in dogs. Blood flow was measured and liver structure and function were studied. Experiments with 7 series of dogs included organ removals (splenectomy, pancreatectomy) and shunts (splenocaval derivation, mesenterico-caval shunt, spleno-mesenterico-caval shunt). Five to 8 dogs were used in each series for a total of 68 dogs. Hepatic biochemical tests, determination of hepatic blood flow (by ^{198}Au, ^{90}Kr, Bengal Rose, and Indocyanine Green) and serial liver biopsies before and after surgery (and in the long survivals) were performed. Results show a flow decrease of 32.3% and 20.6% after mesenterico and spleno-cava derivations. Histologically, a swollen cytoplasm was encountered (probably due to this important flow decrease); but no modifications of the cells (atrophy) nor of their fat and glycogen content were observed. After pancreatectomy there was a flow decrease of 14.2% and liver cord atrophy, bile retention, important fatty degradation, and disappearance of glycogen were seen. All of these modifications were avoided by giving insulin s.c. after pancreatectomy. These results suggest that graft atrophy after auxiliary liver transplantation is due in part to an insufficient supply of insulin.

1757 EFFECTS OF ETHANOL ON HEPATIC MICROSOMAL DRUG-METABOLIZING ENZYMES IN THE RAT. *(E.)* Liu, S. J.; Ramsey, R. K.; Fallon, H. J. (Univ. North Carolina Sch. Med., Chapel Hill). *Biochem. Pharmacol.* 24(3):369-378, 1975.

The activities of several hepatic microsomal drug-metabolizing enzymes were determined by various methods in male and female Sprague-Dawley rats after administration of 20% ethanol or an isocaloric amount of glucose in drinking water for 10-49 days. The aniline hydroxylase activity increased 20-72%,

whereas the activities of both pentobarbital hydroxylase and benzphetamine N-demethylase were decreased about 33% in male rats given ethanol (4 g/kg) and killed without ethanol withdrawal. Twenty-four hr after removal of ethanol, the aniline hydroxylase remained elevated but a 30-40% increase of both pentobarbital hydroxylase and benzphetamine N-demethylase above control values occurred. Six days later, all 3 of these microsomal enzymes returned to normal control values. The reduction in pentobarbital hydroxylase and benzphetamine N-demethylase could not be attributed simply to high endogenous ethanol levels since: 1) addition of high concentrations of ethanol *in vitro* inhibited microsomal aniline hydroxylase and pentobarbital hydroxylase but did not reduce benzphetamine N-demethylase; and 2) acute administration of ethanol p.o. by gastric tube, which markedly elevated the blood ethanol level, did not result in a decline in the enzyme activities. These 2 findings suggest the observed decrease in microsomal enzymes in male rats required persistent ethanol exposure. In contrast to male rats, female rats given ethanol for 28 days showed a 50% increase in aniline hydroxylase activity, but the activities of pentobarbital hydroxylase and benzphetamine N-demethylase were not decreased. Moreover, administration of ethanol for 28 days to female rats did not reduce the response of these 3 enzyme activities to pentobarbital administration. It is concluded that the effects of chronic ethanol ingestion on the hepatic microsomal drug-metabolizing enzymes are complex and depend on sex, exposure to other agents and, most importantly, on the duration and proximity of ethanol intake.

1758 EFFECT OF ACUTE ETHANOL ADMINISTRATION ON TRYPTOPHAN OXYGENASE ACTIVITY IN RAT LIVER. *(E.)* Morland, J. (Dep. Pharmacol., Univ. Oslo, Norway). *Acta Pharmacol. Toxicol. (Kbh.)* 35(5): 347-360, 1974.

Tryptophan oxygenase activity was studied in liver homogenates from male Wistar rats following short-term ethanol administration. Ethanol was given in the following ways: 1) p.o. mixed with sucrose in drinking water *ad lib* for 1 or 18-21 hr; 2) i.p. as a single dose (2.5 or 3.3 g/kg). Short-term ethanol treatment was also given to animals which had received prior ethanol treatment for 3-4 weeks. Ethanol was added in some studies to perfusates of isolated livers (final concentration 0.2%). Although p.o. ethanol caused only a slight increase in tryptophan oxygenase activity and no change in plasma corticosterone levels, i.p. injection produced a 337% rise in liver enzyme activity and about a 400% increase in plasma corticosterone concentration. Cycloheximide (1.5 mg/kg) reversed the increase in tryptophan oxygenase activity following i.p. ethanol injection. Ethanol had no effect on basal enzyme activity of perfused liver but did slightly enhance induction by dexamethasone. Degradation of tryptophan oxygenase in perfusates following cycloheximide administration (20 µg/ml) was unaffected by ethanol. I.p. injection of ethanol into rats pretreated with ethanol produced a 268% increase in hepatic enzyme activity, a value less than that for normal rats. Tryptophan oxygenase activity in ethanol-pretreated rats was significantly reduced following p.o. ethanol ingestion. The acute stimulatory effect of ethanol on tryptophan oxygenase activity of the rat thus appears to be due to a rise in plasma corticosterone which induces enzyme synthesis.

1759 A POSSIBLE MECHANISM FOR THE PEROXIDATION OF LIPIDS DUE TO CHRONIC ETHANOL INGESTION. *(E.)* Reitz, R. C. (Dep. Biochem., Univ. North Carolina, Chapel Hill). *Biochim. Biophys. Acta* 380 (2):145-154, 1975.

The effects of chronic ethanol ingestion on NADPH-oxidase and on the NADPH-catalyzed peroxidation of lipids in rat liver microsomes were studied. Male Buffalo rats were maintained for 1 month on carefully controlled diets of Purina Lab Chow (41-67 kcal/day), with 1 group receiving 20% ethanol (32% of the total daily intake of calories) as the only source of drinking water and another group receiving an isocaloric amount of glucose in the drinking water. The intake of vitamin E was 0.8-1.2 mg/day. Chronic ethanol ingestion resulted in an increase in the activity of microsomal NADPH oxidase; the increase was not due to major variations in protein or RNA concentrations. In addition, the ethanol-treated animals had about 1.5 times the rate of formation of malondialdehyde as did the controls; this difference was statistically significant. In the control rats, there was a lag before the formation of malondialdehyde became linear with time; chronic ethanol ingestion shortened this lag. Compared with controls, the energy of activation for the formation of malondialdehyde was significantly elevated in ethanol-treated animals. Chronic ethanol ingestion enhanced the ability of the microsomal fraction to consume oxygen when incubated in the lipid peroxidation system. It also resulted in significant changes in the composition of the polyenoic fatty acids of the microsomes: arachidonate decreased, while linoleate increased. The C_{22} polyenes showed a nonsignificant decrease. Ethanol ingestion appeared to induce changes in the microsomal membranes which resulted in additional or alternate, possibly abnormal, pathways for lipid peroxidation becoming functional. The data also indicate that ethanol ingestion enhanced the production of peroxides *via* the microsomal-catalyzed oxidation of NADPH.

1760 DISTRIBUTION OF ZINC IN SKELETAL MUSCLE AND LIVER TISSUE IN NORMAL AND DIETARY CONTROLLED ALCOHOLIC RATS. *(E.)* Wang, J.; Pierson, Jr., R. N. (St. Luke's Hosp. Cent., Columbia Univ., New York, N.Y.). *Lab. Clin. Med.* 85(1):50-58, 1975.

The compartmental distribution of zinc in the intracellular components of normal and alcoholic rats was studied, along with the relationships between serum and tissue zinc levels and the correlation between these parameters and the development of alcoholism. Five 6-week-old male Sprague-Dawley rats were given laboratory chow and 20% ethanol as the sole source of drinking water; controls were given tap water. After 2, 5, 9, or 14 weeks, the animals were killed and the zinc levels in tissue

measured by atomic absorption spectroscopy. Zinc levels in tissues of control rats remained stable at 108 µg/ml (plasma), and 125 (muscle), 30.3 (muscle cell sap), 70 (muscle cell sap-free fraction), 209 (muscle mitochondria), 198 (liver), 125 (liver cell sap), 79.5 (liver cell sap-free fraction), and 291 µg/g dry tissue (liver mitochondria). The ethanol-fed rats showed marked decreases in all liver fractions after 2 weeks, the value being 120 µg/g in the total liver tissue after 2 weeks. The greatest decrease (to 35% of control values) was seen in the liver mitochondria. The zinc levels in the muscle tissue and plasma showed monotonic gradual declines at the rate of 3% week, become statistically different from control values after 9 weeks. The control rats showed normal weight gain, while the ethanol-fed rats gained at a rate of 52% control values up to 5 weeks, showing no subsequent gain and weighing only 62% of control values at 14 weeks. The results indicate that, although the liver mitochondria are significantly more affected by plasma alcohol than muscle mitochondria, the zinc-depleting effects of alcohol are not specific for the liver.

1761 ANALYTICAL STUDY OF MICROSOMES AND ISOLATED SUBCELLULAR MEMBRANES FROM RAT LIVER. IV. BIOCHEMICAL, PHYSICAL, AND MORPHOLOGICAL MODIFICATIONS OF MICROSOMAL COMPONENTS INDUCED BY DIGITONIN, EDTA, AND PYROPHOSPHATE. *(E.)* Amar-Costesec, A.; Wibo, M.; Thines-Sempoux, D.; Beaufay, H.; Berthet, J. (Physiol. Chem. Lab., Univ. Louvain, Belgium). *J. Cell Biol.* 62(3):717-745, 1974.

Rat liver microsomal constituents were classified according to physical properties and cytological location after treatment with digitonin, pyrophosphate and EDTA. Homogenates from 0.27 g fresh weight of liver/ml were fractionated in 0.25 M sucrose (pH 7.4) and the resulting pellets treated with either 0.235% (weight/volume) digitonin, 15 mM sodium pyrophosphate (pH 8.2), 50 mM EDTA (pH 7.4) or with 0.235% (weight/volume) digitonin after centrifugatin of EDTA-treated (50 mM) pellets. Microsomes were centrifuged at an integrated force of 3 or 9 x 10^{10} $rad^2 \cdot s^1$ before isopycnic equilibration or differential sedimentation, resp. Enzyme activities were unaffected by the various treatments, except that EDTA caused partial inactivation of 5'-nucleotidase. Digitonin did not affect the sedimentation properties of any of the constituents nor did it alter the morphology of the microsomes except that a few large smooth elements appeared broken or fenestrated as seen by electron microscopy. Digitonin significantly increased the median densities of the constituents in the group classified as *a2* (5'-nucleotidase, alkaline phosphodiesterase I, alkaline phosphatase, and cholesterol from 0.028 to 0.035 density units) and group *a3* (galactosyltransferase 0.014 units), but did not affect the other microsomal constituents. Pyrophosphate and EDTA detached a substantial part of RNA and released a significant amount of protein. Both also decreased the densities of the microsomal constituents in group *a2* slightly, and greatly decreased the densities in group *b* (NADH cytochrome c reductase, NADPH cytochrome c reductase, aminopyrine demethylase, cytochrome b_5 and P 450) and group *c* (glucose 6-phosphatase, esterase, nucleoside diphosphatase, β-glucuronidase and glucuronyltransferase). The combination of EDTA and digitonin increased the mean density of group *a2* compared with that of groups *b* and *c*. Dense subfractions showed mainly broken appearing vesicles after treatment with the combination under the electron microscope. It is suggested that the microsomal constituents in group *a1* (monoamine oxidase) are located in the outer membranes of mitochondria, those in group *a2* in the plasma membranes, those in *a3* in the Golgi complex, those in both groups *b* and *c* in the smooth and rough endoplasmic reticulum, and RNA and ribosomes in the rough endoplasmic reticulum. In conclusion, the behavior of the constituents after treatment with digitonin, EDTA, and pyrophosphate confirmed the classification from previous studies of rat liver microsomal constituents.

1762 PERINATAL DRUG METABOLISM AND MORPHOLOGICAL CHANGES IN THE HEPATOCYTES OF NORMAL AND PHENOBARBITAL-TREATED GUINEA PIGS. *(E.)* Kuenzig, W.; Kamm, J. J., Boublik, M.; Jenkins, F.; Burns, J. J. (Roche Inst. Mol. Biol., Nutley, N.J.). *J. Pharmacol. Exp. Ther.* 191(1):32-44, 1974.

Microsomes were isolated from the livers of guinea pig fetuses delivered by cesarean section days 33, 45, 53, and 63 of gestation and from the livers of neonatal and adult guinea pigs. Benzo(a)pyrene, *p*-chloro-N-methylaniline, and chlorcyclizine were used as substrates to measure the rate of development of drug metabolic activity. Some animals were subjected to acute phenobarbital pretreatment by the administration of sodium phenobarbital to pregnant dams for 3 days at a dose of 100 mg/kg in the drinking water (1 mg/ml); the animals drank about 10 ml/day/100 g. Phenobarbital was administered chronically at a dose of 30 mg/kg daily in the drinking water (0.3 mg/ml) for 14 days from day 49 through day 62 of gestation. The oxidation of the drug substrates and the rate of reduction of cytochrome P-450 increased slowly during the fetal life of the guinea pig whereas the concentration of cytochrome P-450 in the liver increased rapidly. Forty-eight to 72 hr after birth, substrate oxidation and P-450 reduction occurred at a rate equal to or greater than the rate observed in preparations from adult animals, but the concentration of cytochrome P-450 was slightly below adult levels. Acute sodium phenobarbital pretreatment stimulated the hepatic benzo(a)pyrene hydroxylase activity in the fetus through the second half of gestation (days 33-63). *p*-Chloro-N-methylaniline demethylase and cytochrome P-450 reductase activities, as well as cytochrome P-450 levels, were not induced by phenobarbital treatment of the dam until just prior to parturition (day 63). The ultrastructure of the hepatocyte of the developing guinea pig was examined by electron microscopy and a direct correlation between microsomal mixed-function oxidase activity and the proliferation of the smooth endoplasmic reticulum was observed in normal and phenobarbital-treated animals. The results of this investigation are consistent with the hypothesis that factors which control the proliferation of the smooth endoplasmic reticulum are also important in controlling

the level of hepatic microsomal mixed-function oxidation.

1763 RATE-LIMITING STEPS IN DRUG METABOLISM BY MICROSOMES FROM CCl_4-CIRRHOTIC RAT LIVER. *(E.)* Tsyrlov, I. B.; Lyakhovich, V. V. (Acad. Med. Sci. USSR, Siberian Branch, Novosibirsk). *Chem. Biol. Interact.* 10(2):77-89, 1975.

The substrate-binding capacity of microsomal cytochrome P-450, the activity of the enzymes in the NADPH-dependent electron-transport chain, and other possible rate-limiting factors in determining the overall rate of hydroxylation reactions by the microsomal fraction of the cirrhotic liver was studied. Histological symptoms of subacute toxic hepatitis and cirrhosis were produced in male Wistar rats following 3-6 weeks of biweekly periods of poisoning by CCl_4 inhalation. The development of CCl_4-cirrhosis was accompanied by a decrease in the metabolizing activity of type-I (aminopyrine, hexobarbital), type-II (aniline, metyrapone), and "modified type-II" (corticosterone) substrates in liver microsomes. Cytochrome P-450 content also appeared to decrease during chronic CCl_4 intoxication. The hydroxylation activity of the type-II and "modified type-II" substrates during the period of cirrhotic development was determined by the P-450 content and the amplitude of maximal spectral changes observed in the presence of excess substrate. Type-I substrate metabolism, however, was limited in the precirrhotic phase by the state of the "substrate-binding protein" contained in P-450 as well as by the activity of NADPH-cytochrome P-450 reductase. On the other hand, the *N*-demethylating activity in the cirrhotic microsomes did not depend on the concentration of P-450, on the amplitude of the maximal spectral changes, or on the K_s value. The data suggest that NADPH-cytochrome P-450 reductase activity is a rate-controlling reaction, in the metabolism of aminopyrine by cirrhotic liver microsomes.

1764 LIPID PEROXIDATION IN ISOLATED HEPATOCYTES. *(E.)* Hogberg, J.; Orrenius, S.; Larson, R. E. (Dep. Forensic Med., Karolinska Inst., Stockholm, Sweden). *Eur. J. Biochem.* 50(3):595-602, 1975.

Studies were conducted to determine whether peroxidation of intracellular lipids could be induced by addition of ADP-complexed ferric iron to hepatocytes isolated from male Sprague-Dawley rat livers perfused with a collagenase-hyaluronidase solution. Both the production of malondialdehyde (thiobarbituric-acid-reacting substances) and of conjugated dienes was dependent on the ADP-Fe^{3+} concentration (10-80 μM) in a dose-related fashion. Malondialdehyde formation stopped spontaneously within 20 min after the initiation of the reaction and the plateau reached was also related to the ADP-Fe^{3+} concentration. Control experiments revealed that more than 90% of the malondialdehyde accumulating during the incubation period could be ascribed to intracellular production. The cellular NADPH/NADP ratio was always high and only slightly decreased upon ADP-Fe^{3+}-induced lipid peroxidation. The rate of accumulation of malondialdehyde as well as the final level reached during ADP-Fe^{3+}-initiated lipid peroxidation was increased about 30% by the addition of chloral hydrate (10 mM). This apparent stimulatory effect could, however, be ascribed to the inhibition of the mitochondrial oxidation of the malondialdehyde formed during cellular lipid peroxidation, thus allowing more malondialdehyde to accumulate during the process. ADP-Fe^{3+}-induced cellular lipid peroxidation was associated with a 42% decrease in the concentration of glutathione. Also, lowering of the intracellular glutathione level by the addition of diethyl maleate or by simply preincubating the hepatocytes (up to 50 min) promoted the ADP-Fe^{3+} malondialdehyde production, observed even at ADP-Fe^{3+} concentrations which were too low to induce measurable lipid peroxidation in fresh hepatocytes. It is thus concluded that glutathione has an important role in the cell defense against lipid peroxidation. It is further suggested that the isolated hepatocytes provide a suitable experimental model system for the characterization of this and other possible cellular defense mechanisms and how they are affected by the nutritional status of the donor animal.

1765 AGE-RELATED CHANGES IN LIPID PEROXIDATION IN RAT LIVER MITOCHONDRIA. *(E.)* Barrett, M. C.; Horton, A. A. (Dep. Biochem., Univ. Birmingham, England). *Biochem. Soc. Trans.* 3(1):124-126, 1975.

Lipid peroxidation by means of the thiobarbituric acid reaction was studied in freshly isolated rat liver mitochondria and mitochondria treated with ascorbate. The mitochondria were isolated from the livers of female Wistar rats allowed food and water *ad libidum*. As indicated by the concentrations of malondialdehyde in the mitochondria, the peroxidation potential increased about 2.3-fold as the age of the animal increased from 3-24 months; the peroxidation measured in freshly isolated mitochondria was small and fell with age after 6 months. The increase in peroxidation potential may suggest an age-related increase in the degree of polyunsaturation of the mitochondrial lipids. Mitochondrial lipids were assayed for the degree of unsaturation by an iodometric titration; cholesterol contributed less than 1% of the double bonds measured. Little change was noted in the cholesterol:protein ratio of mitochondria with increasing age of the animal. The total degree of unsaturation of the mitochondrial lipids fell in the 3-12-month-old rats and then rose in the 12-24-month-old animals. The discrepancy between this relationship and the observed rise in peroxidation potential with age may be explained in terms of an age-linked increase in the proportion of polyunsaturated fatty acids and a decrease in the proportion of mono-unsaturated fatty acids in the mitochondrial lipids. The observed trends were not due to survival factors in individual animals.

1766 STUDIES ON THE SYNTHESIS AND INTRACELLULAR TRANSPORT OF LIPOPROTEIN PARTICLES IN RAT LIVER. *(E.)* Glaumann, H.; Bergstrand, A.; Ericsson, J. L. E. (Sabbatsberg Hosp., Karolinska Inst., Stockholm, Sweden). *J. Cell Biol.* 64(2):356-377, 1975.

Lipoprotein particles (density <1.03 g/ml) isolated from microsomes from smooth and rough endoplasmic reticulum and from the Golgi apparatus of rat liver were characterized chemically and morphologically. Rough endoplasmic reticulum particles were protein rich (50%) and contained smaller amounts of phospholipids and triglycerides. Smooth particles and especially those from the Golgi were rich in triglycerides and phospholipids and resembled very low density lipoproteins. The rough particles were 200-800 Å in diameter (mean = 420 Å); the smooth particles 200-900 Å (520 Å); Golgi particles 350-950 Å (580 Å); and very low density lipoproteins 300-800 Å (450 Å). In general, lipoprotein particles were rare in the rough endoplasmic reticulum, frequent but diffusely situated in the smooth endoplasmic reticulum, and occurred mainly in clusters in "secretory vesicles" of the Golgi complex. They were rarely seen in the cisternal compartments of the Golgi complex. Following injection of ^{3}H-glycerol, label was initially (15 min) seen in triglycerides and later primarily in phospholipids. Three intracellular triglyceride pools were identified: 1) a cytoplasmic pool with a slow turnover rate; 2) a membrane-associated pool and; 3) a pool corresponding to the triglyceride moiety of the lipoprotein particles, which had the highest initial rate of labeling and fastest turnover. Pulse labeling with ^{3}H-glycerol showed peak labeling at 6 min in both smooth and rough microsomal lipoproteins followed at 10-20 min by peak labeling of the Golgi lipoproteins and finally, after an additional 10-15 min, peak labeling of very low density lipoproteins in serum. It is concluded that the assembly of the apoproteins and lipid moieties into lipoprotein particles, the putative precursors of liver very low density lipoproteins, begins in the rough and continues in the smooth endoplasmic reticulum with discharge into the circulation from the Golgi apparatus.

1767 GROWTH HORMONE STIMULATION OF AMINO ACID TRANSPORT AND UTILIZATION BY THE PERFUSED RAT LIVER. *(E.)* Jefferson, L. S.; Schworer, C. M.; Tolman, E. L. (Milton S. Hershey Med. Cent., Hershey, Pa.). *J. Biol. Chem.* 290(1):197-204, 1975.

The effects of growth hormone, both *in vivo* and *in vitro*, on amino acid transport and utilization were studied in perfused livers from normal and hypophysectomized male Wistar rats. Amino acid transport was studied using 2 isotopically-labeled non-metabolizable amino acid analogues, α-aminoisobutyric acid and 1-aminocyclopentane carboxylic acid (cycloleucine). When normal or hypophysectomized animals were given α-aminoisobutyric acid cycloleucine 24 hr before sacrifice, steady-state distribution ratios between liver and plasma were 4.6 and 5.6, resp., for α-aminoisobutyric acid and from 1 to 2 in both cases for cycloleucine. Treatment with bovine growth hormone (1 mg/100 g, i.v.) 1 hr prior to sacrifice increased the steady-state distributions for both analogues by 19% in normal livers and by 46-48% in livers from hypophysectomized rats. Treatment with growth hormone *in vivo* followed by *in vitro* perfusion with α-aminoisobutyric acid or cycloleucine produced increases in the steady-state distribution ratios which were apparently comparable to those seen *in vivo*. Growth hormone treatment increased the maximum velocity of α-aminoisobutyric acid and cycloleucine accumulation by over 2-fold while only slightly increasing the substrate concentration necessary for half-maximal transport velocity. *In vitro* studies in which livers from hypophysectomized rats were perfused with a non-recirculating medium containing cycloleucine and growth hormone (1 μg/ml) showed a 30% increase in the distribution ratio, indicating that growth hormone was exerting a direct effect in stimulating amino acid accumulation. In the same *in vitro* system, growth hormone also produced: 1) a 23% increase in the rate of incorporation of ^{14}C-valine into liver protein; 2) a 2-fold increase in the rate of gluconeogenesis as determined from the rate of conversion of ^{14}C-amino acids to ^{14}C-glucose; 3) a 40-50% increase in urea production; and 4) 40-250% increases in the intracellular concentrations of threonine, serine, proline, glycine, alanine, lysine and arginine. The last finding indicated that the effects of growth hormone on the first 3 were due to its stimulation of amino acid transport. The time required for a significant effect of growth hormone on amino acid metabolism in the perfused liver was 20-25 min. These data thus confirm and extend previously published findings of stimulated hepatic amino acid transport *in vivo* in growth hormone-treated rats.

1768 VASOACTIVE INTESTINAL POLYPEPTIDE: INACTIVATION IN LIVER AND POTENTIATION IN LUNG OF ANESTHETIZED DOGS. *(E.)* Kitamura, S.; Yoshida, T.; Said, S. I. (Univ. Texas Southwest. Med. Sch., Dallas). *Proc. Soc. Exp. Biol. Med.* 148(1):25-29, 1975.

The role of the liver and lung in the inactivation or potentiation of the biological activity of the vasoactive intestinal polypeptide was examined in 5 anesthetized mongrel dogs. Infusions of the peptide were made into the right ventricle, the left ventricle, and the portal vein; each animal received 4 doses, ranging from 0.29-2.23 μg/kg. Systemic arterial blood pressure, tidal volume, breathing frequency, and minute ventilation were measured continually. The biological effects of the peptide were measured in terms of decreases in arterial blood pressure and respiratory stimulation. The peptide elicited a hypotensive response which became more pronounced with increasing dosage. For any given dose, the magnitude of the hypotension was always smallest and its duration shortest after infusions into the portal vein. Except for the smallest dose, infusions of the peptide into the right or left ventricle were always followed by an increase in minute ventilation. However, the mean respiratory stimulation in all 5 dogs following portal infusion was significantly smaller than that resulting from right ventricular infusion. Infusions into the right ventricle were somewhat more potent than infusions into the left ventricle. The apparent inactivation of vasoactive intestinal polypeptide during passage through the liver implies that its actions may be confined to the gastrointestinal tract, the liver, and the portal circulation. The increase in biological activity of the

peptide during passage through the lung may be attributable to the release of additional vasodilator substances or to further activation of the peptide.

1769 SERUM ANTITRYPSIN SYNTHESIS BY THE ISOLATED PERFUSED RAT LIVER. *(E.)* Rowley, P. T.; Miller, L. L. (Univ. Rochester Sch. Med. Dent., New York). *Proc. Soc. Exp. Biol. Med.* 148(1):145-150, 1975.

The isolated perfused rat liver was studied to establish the liver as the site of serum antitrypsin synthesis. Although the pattern of antitrypsin activity of normal rat serum differs from that of human serum, both show the greatest activity/mg protein in the α-globulin fraction. When a liver from a normal rat was perfused for 12 hr with a medium containing glucose and amino acids, the antitrypsin activity rose steadily throughout. Cortisol plus insulin caused no change in the rate of synthesis of antitrypsin activity over a 24-hr period. The isolated perfused rat liver appeared to synthesize antitrypsin at 10 times its *in vitro* rate. The antitrypsin activity of the rat perfusate resembled that of normal human plasma in regard to heat lability (loss of 45% of its activity in 5 min at 56 C), acid lability (loss of activity below pH 6), and apparent molecular size as determined by elution on Sephadex G-200 columns. Starch block electrophoresis revealed 2 peaks of antitrypsin activity in the rat liver perfusate: a broader peak at the trailing edge of the α_1-globulin region; and a sharper peak in the β-globulin region. The data suggest that in the rat, the liver synthesizes material with properties similar to those of human α_1-antitrypsin at a rate compatible with the liver as the principle site of synthesis.

1770 EXTRAHEPATIC DISTRIBUTION OF SULFOBROMOPHTHALEIN. *(E.)* Klaassen, C. D. (Univ. Kansas Med. Cent., Kansas City). *Can. J. Phys. Pharm.* 53(1): 120-123, 1975.

The body tissue distribution and biliary excretion of BSP was studied in male Sprague-Dawley rats. When BSP was administered i.v. to rats at four different doses (18.75, 37.5, 75, and 150 mg/kg), less than 0.5% of each dose was excreted into the urine and between 70 and 85% was excreted into the bile within 6 hr after administration. When the amount of BSP was measured in plasma, liver and bile 10 min after the i.v. administration of either a high (150 mg/kg) or a low (18.75 mg/kg) dose of BSP, only 60% of the dose was accounted for. The concentration of BSP and ^{125}I-labeled albumin was measured in various tissue samples 10 min after administration of 17.5 or 150 mg of BSP or [^{125}I]albumin/kg. More BSP was found in all tissues than was contained in the plasma. Thus, the distribution of BSP is not limited to the liver and plasma. It is concluded that during excretion, BSP leaves other tissues (kidney, spleen, lung, etc.) and is ultimately excreted into the bile.

1771 THE PROPERTIES OF HYDROGEN PEROXIDE PRODUCTION UNDER HYPEROXIC AND HYPOXIC CONDITIONS OF PERFUSED RAT LIVER. *(E.)* Oshino, N.; Jamieson, D.; Chance, B. (Sch. Med., Univ. Pennsylvania, Philadelphia). *Biochem. J.* 146(1):53-65, 1975.

The properties of H_2O_2 production were examined in the hemoglobin-free, non-circulatory perfused livers of male Holzman rats. H_2O_2 production was determined by spectrophotometric measurement of the catalase-H_2O_2 intermediate. The H_2O_2 production with 1 mM lactate and 0.15 mM pyruvate was 82 nM/min/g liver or 333 nM/min/100 g body weight in the liver of fed rats at 30 C. This rate decreased to almost half in the livers of starved and phenobarbital-pretreated rats. When H_2O_2 production was stimulated by urate infusion, (0-100 μM), almost all of the H_2O_2 produced by the uricase reaction was decomposed by the catalase reaction. During the demethylation reaction of aminopyrine, no change in H_2O_2 production was detected by the present method. Thus, microsomal H_2O_2 production observed in isolated subcellular fractions appeared not to contribute significantly to H_2O_2 production in the whole organ. Whereas the rate of the glycolate-dependent H_2O_2 was halved at an intracellular O_2 concentration that caused a 10% increase in the reduction state of cytochrome c, the half-maximal rate of H_2O_2 production with lactate and pyruvate was observed at an O_2 concentration that caused a 40% increase in the reduction state of cytochrome c in the liver. No further increase in the rates of H_2O_2 production was obtained by increasing O_2 pressure up to 5 x 10^5 ambient pressure. The rate of ethanol oxidation through the catalase 'peroxidatic' reaction varied, depending on the substrate availability. The maximal capability of this pathway in ethanol oxidation reached approximately 1.5 μM/min/g liver, when a mixture of urate, glycolate and octanoate was infused to enhance H_2O_2 production. These studies indicate that endogenous H_2O_2 production detected in the perfused liver is most likely of mitochondrial origin, that the primary events associated in the O_2-toxicity phenomenon are not produced in the H_2O_2 producing reactions of the perfused liver, and that this microsomal H_2O_2 producing system has no significant physiological role in ethanol metabolism in this system.

1772 INDUCTION OF CYSTATHIONASE IN HUMAN FOETAL LIVER. *(E.)* Heinonen, K.; Raiha, N. C. R. (Dep. Med. Chem., Univ. Helsinki, Finland). *Biochem. J.* 144(3):607-609, 1974.

Liver explants from second-trimester human fetuses were incubated with dexamethasone, glucagon, or dibutyryl cyclic AMP plus theophylline to determine whether cystathionase can be induced in organ culture. Explants less than 1 mm^3 were placed on wire grids at the liquid gas interphase and preincubated for 24 hr before addition of the agents. Eagle's minimum essential medium with Hank's balanced-salt solution containing twice the normal concentration of glucose, glutamine and bicarbonate (pH 7.6) was used. No cystathionase activity was found in fresh specimens, but a post-mortem sample from a full-term infant showed 70 nM cysteine/60 min/mg protein. Also, no cystathionase activity was found in fetuses

30-50 mm, and only trace amounts in those 90-170 mm after 48 hr incubation. Addition of pyridoxal 5'-phosphate (1-100 μg/ml) did not increase the activity of cystathionase in any of the fetuses. Dibutyryl cyclic AMP (0.2 mM) plus theophylline (0.5 mM) significantly increased the activity of cystathionase to 192 nM/60 min/mg protein compared with controls (31 nM/60 min/mg protein). This activity was significantly more than than that produced by either agent alone at the same or 10-fold concentrations. Dexamethasone (60 μg/ml) and glucagon (100 μg/ml) produced similar effects. Simultaneous incubation with cycloheximide (10 μg/ml) or actinomycin D (30 μg/ml) abolished the effects of the inducing agents. The activity of cystathionase could not be stimulated in the explants from the smallest (early first-trimester) fetuses. This suggests that protein synthesis is required for the increased enzyme activity; thus, a certain developmental stage must be reached before fetal liver tissue has this competence to respond to externally administered pharmacological agents. It is concluded that cystathionase activity can be induced in second-trimester human fetal liver *in vitro*.

1773 STUDIES ON BILIRUBIN AND STEROID GLUCURONIDATION BY RAT LIVER MICROSOMES. *(E.)* Jacobson, M. M.; Levin, W.; Conney, A. H. (Dep. Biochem. Drug Metab., Hoffman-La Roche, Inc., Nutley, N.J.). *Biochem. Pharmacol.* 24(6):655-662, 1975.

The effects of *in vivo* phenobarbital administration and the *in vitro* addition of detergents on the glucuronidation of bilirubin and several steroids by rat liver microsomes were studied. In short term experiments, sodium phenobarbital (75 mg/kg/day) was administered i.p. for 3-4 days to male and female Long Evans rats. In long term experiments, the drug was administered in the drinking water to male and female Long Evans rats (175 mg/kg/day) and male and female homozygous Gunn rats (100 mg/kg/day) for 20 days. The addition of digitonin, cholate, Triton-X-100, Emulgen 911, Triton N-101, and deoxycholate to the microsome preparations from phenobarbital-treated and control rats increased the glucuronidation of bilirubin, testosterone, estradiol, estrone, and estriol. The extent of activation by digitonin and the concentration causing maximal activation were substrate dependent. There was a partial requirement for Mg^{++} in steroid glucuronidation. Short-term treatment with sodium phenobarbital increased the glucuronidation of testosterone and estradiol/mg of protein but had no effect on estrone glucuronidation. Administration of the drug for 20 days to Long Evans rats increased bilirubin, testosterone, and estrone glucuronidation/mg protein, but had little or no effect on estradiol and estriol glucuronidation. However, when UDP-glucuronyl transferase activity was calculated/g liver (wet weight), an increase in the glucuronidation of estriol and estradiol was seen, resulting from the large stimulatory effect of phenobarbital on the synthesis of total microsomal protein. Liver microsomes from Gunn rats, deficient in the ability to conjugate bilirubin, did conjugate testosterone, estradiol, estrone, and estriol. Digitonin increased the glucuronidation of testosterone but not that of estrone; sodium phenobarbital stimulated microsomal steroid glucuronidation to varying degrees. The data suggest that there are several glucuronyl transferases in rat liver microsomes, and that the glucuronyl transferase for bilirubin is different from the enzyme(s) which metabolizes testosterone, estradiol, estrone, and estriol by glucuronidation.

1774 EXTRAHEPATIC BILIRUBIN FORMATION AND CONJUGATION IN THE DOG. *(E.)* Royer, M.; Noir, B. A.; Sfarcich, D.; Nanet, H. (Dr. A. Posadas Gen. Hosp., Haedo, Argentina). *Digestion* 10(6):423-434, 1974.

The site of formation and distribution of bilirubin formed outside the liver in the dog was studied, along with the chemical nature of the pigment conjugated in nonhepatic tissues. Total hepatectomy, hepatectomy plus splenectomy or enterectomy, or hepatectomy plus splenectomy and enterectomy or bilateral nephrectomy were performed on male and female mongrel dogs. Some of the hepatectomized dogs were injected i.v. with 2 or 10 mg/kg crystalline bilirubin within 15 min after removal of the liver. The animals remained unconscious throughout the study, and blood and urine were collected at 3-hr intervals from those which remained alive for 15 hr or more. After total hepatectomy, increased bilirubin concentrations were found in the circulating plasma and bound in the lung, kidney, small intestine, omentum, and/or heart. Small amounts of the accumulated pigment were also excreted in the urine. Both unconjugated and direct-reacting serum bile pigments increased significantly, especially in the dogs injected with bilirubin. Analysis of the bile pigments in the serum and urine of the hepatectomized animals indicated that approximately 10% of the conjugates were present as bilirubin xyloside and 25% were present as bilirubin glucoside. The results in the enterectomized, splenectomized, and nephrectomized dogs suggested that the spleen, intestine, and kidney may be sites of bilirubin formation and conjugation in the hepatectomized dog. These observations are consistent with the existence of some glucuronyl transferase activity in the kidney and gastrointestinal mucosa.

1775 BILIRUBIN BINDING TO HEPATIC Y AND Z PROTEIN (LIGANDIN): TISSUE BILIRUBIN CONCENTRATIONS IN PHENOBARBITAL TREATED GUNN RATS. *(E.)* Davis, D. R.; Yeary, R. A. (Dep. Vet. Physiol. Pharmacol., Ohio State Univ. Columbus). *Proc. Soc. Exp. Biol. Med.* 148(1):9-13, 1975.

The effects of phenobarbital treatment on the tissue distribution of bilirubin as affected by bilirubin binding to cytoplasmic liver proteins Y and Z were studied in the icteric Gunn rat. Gunn and Sprague-Dawley rats received daily i.p. injections of sodium phenobarbital (80 mg/kg) or saline (control) for 7 or 14 days. After 7 days, the liver UDP-glucuronyltransferase activity was measured, and after 14 days, the plasma bilirubin levels were determined. In another study, Gunn rats were injected i.v. with ^{14}C-bilirubin after 4 or 14 daily injections of sodium phenobarbital. Sodium phenobarbital treatment for 4 days significantly lowered the plasma bilirubin

levels in male and female Gunn rats; the levels tended to return to pretreatment values after 14 days of treatment. Phenobarbital treatment resulted in increased levels of *p*-nitrophenol glucuronidation in heterozygous and homozygous Gunn rats; a considerably greater increase was seen in the Sprague-Dawley rats. After 7 days of phenobarbital treatment, bilirubin glucuronidation increased 260% in the Sprague-Dawley rats, 126% in the heterozygous Gunn rats, and not at all in the homozygous Gunn rats. The major bilirubin binding tissues in the homozygous Gunn rat were the liver, carcass, skin, gastrointestinal tract, and blood. Sodium phenobarbital treatment for 4 days significantly increased the percent of ^{14}C-bilirubin recovered from the liver and significantly decreased the amount recovered from the gastrointestinal tract; phenobarbital treatment for 14 days resulted in more pronounced alterations. Bilirubin binding to both Y and Z proteins was significantly increased with phenobarbital treatment in the 4- and 14-day treatment groups, the increase in binding being more pronounced after 14 days. The data suggest that phenobarbital treatment resulted in the induction of Y protein synthesis in the liver and an associated increased capacity for the uptake and storage of bilirubin by this organ.

1776 EFFECT OF FREE AND CONJUGATED BILE SALTS ON α-AMYLASE ACTIVITY. *(E.)* O'Donnell, M. D.; McGeeney, K. F.; FitzGerald, O. (Dept. Med. Ther., Univ. Coll., Dublin, Ireland). *Enzyme* 19(4): 129-139, 1975.

The effect of individual bile salts on *in vitro* α-amylase hydrolysis of insoluble Cibachron Blue starch was studied at pH 6.0, the average pH in the jejunum. Control tubes contained 100 mM/liter phosphate buffer, 100 mM/liter NaCl, 1.0 mM/liter $CaCl_2$ diluted with water, and 200 mg of blue starch as substrate. Cholic acid, deoxycholic acid, chenodeoxycholic acid, and lithocholic acid were dissolved in NaOH, buffered, and added to the tubes. Stock solutions of the conjugated sodium cholate, taurocholate, taurodeoxycholate, and taurochenodeoxycholate were dissolved in water. With sodium cholate, taurocholate, and taurodeoxycholate, the enzyme activity in the tubes was increased to 150-160% of control values at a bile salt concentration of about 1 mM/liter. The increased activity extended up to 4 mM/liter. Sodium deoxycholate activated the amylase hydrolysis of the starch up to 0.75 mM/liter (150%) and inhibited it at about 2.5 mM/liter; a similar pattern was seen with taurochenodeoxycholate, which stimulated enzyme activity up to 0.25 mM/liter (135%) and inhibited it above 1.5 mM/liter. Chenodeoxycholate and lithocholate exerted marked inhibition at concentrations as low as 0.5 mM/liter, the inhibition of α-amylase by the former being competitive with both soluble and insoluble starch substrates. The data suggest that bile salts may play a central role in the digestion of carbohydrate, fat, and protein, contributing to the many interactions which occur between enzymes and substrates.

1777 METABOLISM AND BILIARY EXCRETION OF ETHACRYNIC ACID. *(E.)* Klaassen, C. D.; Fitzgerald, T. J. (Univ. Kansas Med. Cent., Kansas City). *J. Pharmacol. Exp. Ther.* 191(3):548-556, 1974.

The biliary excretion of ethacrynic acid (EA) was studied quantitatively and the chemical forms in which it is excreted into bile were determined. ^{14}C-Ethacrynic acid was given i.v. to male Sprague-Dawley rats (1 μCi) and male mongrel dogs (5 μCi). The metabolites of EA were separated by thin-layer chromatography. In rats within 4 hr after the i.v. administration (5 or 50 mg/kg) of ^{14}C-EA, 60-70% was excreted into the bile. Less than 25% was EA and the remainder was biotransformation products. Two major metabolites were identified: the glutathione adduct of EA and EA-mercapturate. Dogs excreted 57% of a 5 mg/kg dose into bile within 4 hrs. Less EA was metabolized by the dog than the rat; 38% of the radioactivity excreted into dog bile was in the form of EA. The dog formed a third metabolite, a cysteine adduct of EA. Seventeen percent of the ^{14}C excreted into the bile remained unidentified. It is suggested that EA is actively transported into the bile and that the unmetabolized EA would probably undergo an enterohepatic circulation, that the EA metabolites might be reconverted to the parent compound of digestive enzymes and by intestinal flora and then also undergo enterohepatic circulation.

1778 THE CHOLERETIC ACTION OF SODIUM TYROPANOATE (BILOPAQUE SODIUM) IN THE DOG: PRELIMINARY OBSERVATIONS. *(E.)* Nelson, J. A.; Staubus, A. E.; Amberg, J. R. (VA Hosp., San Francisco, Calif.). *Invest. Radiol.* 9(6):438-443, 1974.

Excretion studies on sodium tyropanoate were performed on 3 unanesthetized, trained, adult dogs (2 male, 1 female, 20-30 kg) with a modified Thomas cannula (inserted 18-28 months earlier) in the duodenum opposite the ampulla of Vater. Each experiment was preceded by a 1-2 hr control period, with sodium taurocholate given at a constant infusion rate of 8 μM/min. An average bile flow of 0.14 ml/kg/15 min was established as a baseline volume for these 3 dogs. Sodium tyropanoate solution was prepared to an osmolarity of 308 mOsm the resulting solution having a pH of 7.2. This contrast medium was given as an i.v. bolus at a dose of 50 mg/kg (identical to the dose of sodium iopanoate given to these same dogs in an earlier study). Bile volume was determined at 15-min intervals and the bile iodine concentration determined using the method of K-shell fluorescent activation analysis. Timed blood samples were also obtained and iodine concentration determined by fluorescent excitation analysis. One week later, each dog was similarly studied with an identical dose of sodium tyropanoate given as a bolus through a tube in the duodenal cannula into the proximal jejunum. The bile volume curves were similar for all 6 sodium tyropanoate experiments. When the data from the sodium tyropanoate experiments were compared with the data from earlier experiments using sodium iopanoate, the gross kinetics of the 2 drugs were found to be quite similar. Sodium tyropanoate, however, caused a choleresis that led to the excretion of a more dilute solution of contrast material in hepatic bile. Crossover studies, which were conducted with

a 2-week interval, indicated that the choleresis was not related to any temporal changes in the animal preparation. The results of these experiments indicate that, while sodium tyropanoate and sodium iopanoate have similar cumulative bile excretion and blood curves, they differ in their effect on bile flow. This difference is reflected by the different concentrations of these 2 agents found in hepatic bile. These experiments do not explain why this difference occurs.

1779 PHARMACOLOGICAL REVERSAL OF CHOLESTASIS-ASSOCIATED DECREASE IN HEPATIC CYTOCHROME P-450. *(E.)* Mackinnon, M.; Simon, F. (Univ. Colorado Med. Cent., Denver). *Biochem. Pharmacol.* 24(6):748-749, 1975.

The potential of the established microsomal-inducing agents, phenobarbital and 3-methylcholanthrene, for reversing the decrease in the components of the hepatic microsomal drug-metabolizing system associated with bile duct ligation and ethinyl estradiol treatment was studied. Cholestasis was produced in male Sprague-Dawley rats by double ligation and transection of the common bile duct or by the administration of ethinyl estradiol (5 mg/kg/day, i.m.). Phenobarbital (80 mg/kg/day) or 3-methylcholanthrene (15 mg/kg/day) was administered i.p. immediately after bile duct ligation or simultaneously with the 1st injection of ethinyl estradiol. Both bile duct ligation and ethinyl estradiol were associated with a significant decrease in the liver microsomal activities of cytochromes P-450 and b_5 and in NADPH cytochrome c reductase. Phenobarbital and 3-methylcholanthrene increased the level of cytochrome P-450, and only phenobarbital increased NADPH cytochrome c reductase. Simultaneous administration of phenobarbital and ethinyl estradiol slightly increased the level of cytochrome P-450, while simultaneous administration of ethinyl estradiol and 3-methylcholanthrene significantly lowered both cytochrome b_5 and reductase. These values did not differ significantly from those seen with ethinyl estradiol alone. When phenobarbital was administered for 3 days following bile duct ligation, the levels of all 3 components increased significantly over those seen after ligation alone. Bile duct ligation, ethinyl estradiol administration, and phenobarbital treatment were associated with a significant decrease in the specific activity of glucose 6-phosphatase. Simultaneous administration of phenobarbital and ethinyl estradiol lowered this activity below levels seen with either agent alone. Thus, the pharmacological reversal of the cholestasis-associated changes in microsomal proteins is specific, only those proteins stimulated by the pharmacological agent being affected.

1780 BILE ACID POOL SIZE IN HAMSTERS DURING GALLSTONE FORMATION AND AFTER CHOLECYSTECTOMY. *(E.)* Bergman, F.; van der Linden, W. (Dep. Pathol.,I Univ. Umea, Sweden). *Z. Ernaehrungswiss.* 13(1/2):37-42, 1974.

To determine whether a decreased bile acid pool size in gallstone patients could be the chief causal factor in the production of lithogenic bile, the pool size was estimated in hamsters on lithogenic and antilithogenic diets before and after cholecystectomy. One group of hamsters was maintained on commercial chow or lithogenic diet 284 for 7-8 days prior to surgical cholecystectomy; another group was cholecystectomized and 10 days later was placed on the commercial or lithogenic diet for 7-8 days. In both the chow-fed and lithogenic diet-fed hamsters, there was a significant decrease in the size of the bile acid pool after cholecystectomy. Before cholecystectomy, the pool size in animals fed the gallstone-inducing diet was larger than that in the chow-fed controls; after cholecystectomy there was no clear difference. Chenodeoxycholic acid constituted a larger part of the pool in the hamsters fed the lithogenic diet than in the controls. The difference in the ratio between the cholic and chenodeoxycholic acid pools was not affected by cholecystectomy. The mean ratio of the pool sizes of the 2 primary bile acids was 1.33 in the animals fed the gallstone-inducing diet and 1.97 in the controls; this difference was statistically significant. The results question whether any pathogenic significance can be ascribed to the diminished bile acid pool found in gallstone patients.

1781 EFFECT OF DIETARY CONJUGATED BILE SALT SUPPLEMENTATION ON BILE ACIDS IN THE SMALL INTESTINE OF THE RAT. CHANGES IN APPARENT FATTY ACID ABSORPTION. *(Fr.)* Demarne, Y.; Sacquet, E.; Van Heijenoort, Y.; Mathis, C. (Natl. Cent. Zootech. Res., Jouy-en-Josas, France). *Ann. Biol. Anim. Biochim. Biophys.* 14(2):239-250, 1974.

For 14 days, adult holoxenic rats were given a diet high in saturated fats (43% palmitic and stearic acids) which was supplemented with 5 parts/thousand of commercial sodium taurocholate. Experimental rats received an average of 123 μM/day bile salts (total 1720 μM). Extracts from the small intestines of experimental rats contained 88 μM bile acids in comparison to 48 μM in the control group. Cholic and deoxycholic acids accounted for 78% and 18%, resp., of bile acids in the controls and for 64% and 22%, resp., of bile acids in rats given the bile salt supplement. β-Muricholic acid was 14% lower and hyodeoxycholic acid 66% lower in experimental rats than in controls. Balance studies revealed that bile salt supplementation affected only lipid utilization. Stool samples contained 7.5% lipids in experimental rats and 15.0% in controls (dry weight). In rats given bile salt supplement, about 60% of the excreted lipids consisted of soluble lipids, while the soluble lipid fraction accounted for only 40% of total lipids in the stools of controls. Since both groups of rats excreted the same quantities of calcium, the high fecal excretion of insoluble lipids by controls cannot be due to decreased calcium utilization or increased excretion of endogenous calcium. Bile salt supplementation in holoxenic rats improved the utilization of long-chain, saturated fatty acids.

1782 PROLYL HYDROXYLASE ACTIVITY IN REGENERATING RAT LIVER. *(E.)* Benjamin, I. S.; Mcgee, J. O'D.; Than, T.; Rodger, M. C.; Blumgart, L. H. (Glasgow R. Infirm., Scotland). *Gut* 15(10):829, 1974.

1783 STIMULATION OF IMMATURE MALE RAT LIVER GROWTH BY PHENOBARBITAL AND 3-METHYLCHOLANTHRENE. *(E.)* Augenlicht, L. H.; Argyris, T. S. (Dep. Biol., Syracuse Univ., N.Y.). *Exp. Mol. Pathol.* 22(1):1-10, 1975.

1784 EFFECTS OF γ-IRRADIATION OF BIOSYNTHESIS OF DIFFERENT TYPES OF RIBONUCLEIC ACIDS IN NORMAL AND REGENERATING RAT LIVER. *(E.)* Markov, G. G.; Dessev, G. N.; Russev, G. C.; Tsanev, R. G. (Inst. Biochem., Bulgarian Acad. Sci., Sofia). *Biochem. J.* 146(1):41-51, 1975.

1785 EFFECT OF PHENOBARBITAL ON LIVER CELL PROLIFERATION FOLLOWING PARTIAL HEPATECTOMY. *(Ger.)* Tuczek, H. V.; Skoruppa, W.; Rabes, H. M. (Pathol. Inst., Univ. Munich, Germany). *Verh. Dtsch. Ges. Pathol.* 54:437, 1973.

1786 BIOLOGICAL ASPECTS OF LIVER TRANSPLANTATION IN THE SWINE. *(Sp.)* Camprodon, R.; Salva, J. A.; Jornet, J.; Guerrero, J. A.; Monasterio, J.; Pineiro, H.; Mendoza, C. G.; Segura, J.; Soley, P.; Canela, M. (Francisco Franco Munic. Hosp., Barcelona, Spain). *Rev. Esp. Enferm. Apar. Dig.* 44(6):531-538, 1974.

1787 HETEROTOPIC AUXILIARY LIVER TRANSPLANTATION IN WISTAR RATS. *(Ger.)* Zelder, O.; Jerusalem, C. R.; Bode, C. (Surg. Clin., Univ. Marburg/Lahn, Germany). *Z. Gastroenterol.* 12(2):103-112, 1974.

1788 MASS ISOLATION AND CULTURE OF RAT KUPFFER CELLS. *(E.)* Munthe-Kaas, A. C.; Berg, T.; Seglen, P. O.; Seljelid, R. (Norsk Hydro's Inst. Cancer Res., Oslo, Norway). *J. Exp. Med.* 141(1):1-10, 1975.

1789 EXPERIMENTAL HEPATIC NECROSIS: MORPHOLOGIC, BIOCHEMICAL AND IMMUNOELECTROPHORETIC STUDIES. *(E.)* Zaki, F. G.; Keysser, C. H. (Squibb Inst. Med. Res., New Brunswick, N.J.). *Am. J. Pathol.* 78(1):11a, 1975.

1790 LASER SURGERY IN EXSANGUINATING LIVER INJURY. *(E.)* Fidler, J. P.; Hoefer, R. W.; Polanyi, T. G.; Bredemeier, H. C.; Siler, V. E.; Altemeier, W. A. (Univ. Cincinnati Med. Cent., Ohio). *Ann. Surg.* 181(1):74-80, 1975.

1791 SOME ASPECTS ON THE TRANSMISSION OF HEPATITIS B ANTIGEN; MODEL EXPERIMENTS BY MOSQUITOES WITH MURINE HEPATITIS VIRUS. *(E.)* Ishii, A.; Yago, A.; Nariuchi, H.; Shirasaka, A.; Wada, Y.; Matuhasi, T. (Inst. Med. Sci., Univ. Tokyo, Japan). *Jpn. J. Exp. Med.* 44(6):495-501, 1974.

1792 AUTORADIOGRAPHIC STUDIES OF RAT SPLEEN AFTER 2/3 HEPATECTOMY. *(Ger.)* Grouls, V.; Yamashita, K.; Helpap, B. (Pathol. Inst., Univ. Bonn, Germany). *Verh. Dtsch. Ges. Pathol.* 58:515, 1974.

1793 STUDIES ON PLASMA MEMBRANES. XXII. FATTY ACID PROFILES OF LIPID CLASSES IN PLASMA MEMBRANES OF RAT AND MOUSE LIVERS AND HEPATOMAS. *(E.)* Van Hoeven, R. P.; Emmelot, P.; Krol, J. H.; Oomen-Meulemans, E. P. M. (Netherlands Cancer Inst., Amsterdam). *Biochim. Biophys. Acta* 380(1):1-11, 1975.

1794 GANGLIOSIDES IN MITOCHONDRIA AND MICROSOMES OF RAT HEPATOMA 27 AND OF RAT LIVER. *(Rus.)* Diatlovitskaia, E. V.; Novikov, A. M.; Gorkova, N. P.; Sorokina, I. B.; Bergelson, L. D. (Shemiakin Inst. Bioorgan. Chem., Moscow, USSR). *Dokl. Akad. Nauk SSSR* 220(4):969-971, 1975.

1795 CYTOSTATIC, CYTOCIDAL AND POTENTIAL ANTITUMOR PROPERTIES OF A CLASS OF QUINOID COMPOUNDS, INITIATORS OF THE DORMANT STATE IN THE SPORES OF Agaricus bisporus. *(E.)* Vogel, F. S.; Kemper, L. A. K.; McGarry, S. J.; Graham, D. G. (Duke Univ. Med. Cent., Durham, N.C.). *Am. J. Pathol.* 78(1):33-48, 1975.

1796 RETICULOENDOTHELIAL FUNCTION AND ENDOTOXEMIA FOLLOWING HEPATIC ISCHEMIA IN THE PRIMATE. *(E.)* Holper, K.; Olcay, I.; Kitahama, A.; Trejo, R. A.; Miller, R.; Brettschneider, L.; Diluzio, N. R.; Drapanas, T. (Sch. Med., Tulane Univ., New Orleans, La.). *Eur. Surg. Res.* 6(1):53, 1974.

1797 THE EFFECTS OF PORTACAVAL SHUNTING AND PORTACAVAL TRANSPOSITION ON SERUM GAMMA-GLOBULIN LEVELS IN THE RAT. *(E.)* Benjamin, I. S.; Ryan, C. J.; McLay, A. L. C.; Horne, C. H. W.; Blumgart, L. H. (Glasgow R. Infirm., Scotland). *Gut* 15(10):825-826, 1974.

1798 PORTACAVAL ANASTOMOSIS IN THE RAT. IV. EFFECT ON LYMPH CIRCULATION. *(It.)* Pettirossi, O.; Cherubini, M.; di Pietrantonio, M.; Lucchetti, P. (Inst. Clin. Surg. Ther., Univ. Trieste, Italy). *Policlinico* [*Chir.*] 80(3):168-174, 1973.

1799 EFFECT OF THE DURATION OF LIGHT EXPOSURE AND TIME OF FEEDING ON CONTROL OF THE CIRCADIAN RHYTHM OF CHOLESTEROL-7α-HYDROXYLASE IN THE RAT. *(Fr.)* Van Cantfort, J. (Lab. Med. Chem., Univ. Liege, Belgium). *C. R. Acad. Sci.* [*D*] *(Paris)* 279:1273-1276, 1974.

1800 STIMULATION OF HEPATIC 3-HYDROXY-3-METHYGLUTARYL CoA REDUCTASE ON ADMINISTRATION OF ATP. *(E.)* Rao, G. S.; George, R.; Ramasarma, T. (Dep. Biochem., Indian Inst. Sci., Bangalore). *Biochem. Biophys. Res. Commun.* 62(3):665-668, 1975.

1801 HYDROXYPYRUVATE AS A GLUCONEOGENIC SUBSTRATE IN RAT HEPATOCYTES. *(E.)* Williamson, D. H.; Ellington, E. V. (Radcliffe Infirm., Oxford, England). *Biochem. J.* 146(1):277-279, 1975.

1802 SPLANCHNIC AND HEPATIC TRIGLYCERIDE SECRETION DURING HYPERCALORIC INTRAVENOUS GLUCOSE INFUSION IN CONSCIOUS SWINE. *(E.)* Wolfe, B. M.; Belbeck, L. W. (Westminster Hosp., London, Canada). *J. Lipid Res.* 16(1):19-27, 1975.

1803 EFFECT OF ETHANOL ON REGENERATION OF PARTIALLY RESECTED RAT LIVER. *(Ger.)* Hauk, H.; Hubner, K. (Senckenberg Cent. Pathol., Clin., Univ. Frankfurt/Main, Germany). *Verh. Dtsch. Ges. Pathol.* 58:525, 1974.

1804 EFFECT OF ETHANOL ON THE FATE OF ADDED OLEATE IN NORMAL, FATTY AND CIRRHOTIC RAT LIVER. *(E.)* Henley, K. S.; Laughrey, E. G. (Univ. Michigan Med. Sch., Ann Arbor). *Gastroenterology* 67(4):796, 1974.

1805 DEMONSTRATION OF THE BIOCHEMICAL MECHANISM INVOLVED IN FATTY LIVER INDUCED IN THE RAT BY PHOSPHORUS: IN VIVO STUDY OF THE METABOLISM OF TRITIATED PALMITIC ACID. *(Fr.)* Truhaut, R.; Claude, J. R.; Thevenin, M.; Podevin, R.; Warnet, J. M. (Teaching Res. Unit Action Mech. Drugs Toxic Comp., Rene Descartes Univ., Paris, France). *Eur. J. Toxicol.* 7(3):167-176, 1974.

1806 MECHANISM OF THE PROTECTIVE ACTION OF SULFONAMIDES IN CCl_4-PRODUCED LIVER INJURIES: THE EFFECT OF SULFONAMIDES ON THE BIOTRANSFORMATION REACTION IN THE RAT LIVER. *(Rus.)* Huen, W. T.; Miuller, D.; Klinger, V. (Inst. Pharmacol. Toxicol., Friedrich Schiller Univ., Jena, Germany). *Farmakol. Toksikol.* 37:176-183, 1974.

1807 CARBON TETRACHLORIDE INDUCED POLYSOME BREAKDOWN. RELATIVE IMPORTANCE OF LIPID PEROXIDATION AND OF BINDING TO RIBOSOMAL COMPONENTS IN THE PROCESS. *(E.)* Castro, J. A.; Diaz Gomez, M. I.; de Castro, C. R.; de Fenos, O. M.; de Ferreyra, E. C.; D'Acosta, N. (Lab. Chem. Bio-Toxicol., CITEFA, Zufriategui y Varela, Argentina). *Res. Commun. Chem. Pathol. Pharmacol.* 10(1):93-104, 1975.

1808 IRREVERSIBLE BINDING OF CARBON TETRACHLORIDE TO MICROSOMAL PHOSPHOLIPIDS. FREE RADICAL NATURE OF THE REACTIVE SPECIE AND ALTERATIONS IN THE PHYSICO-CHEMICAL PROPERTIES OF THE TARGET FATTY ACIDS. *(E.)* Villarruel, M. C.; Castro, J. A. (Lab. Chem. Bio-Toxicol., CITEFA, Zufriategui y Varela, Argentina). *Res. Commun. Chem. Pathol. Pharmacol.* 10(1):105-116, 1975.

1809 SHORT-TERM TOXICITY OF 2,3,7,8-TETRACHLORO-DIBENZO-p-DIOXIN IN THE RAT: STRUCTURAL, ULTRASTRUCTURAL, AND ENZYMOLOGICAL STUDY OF THE LIVER. *(Fr.)* Truhaut, R.; Pham-Huu-Chanh; Van Haverbeck, G.; Azum-Gelade, M. C.; Saint Ruf, G.; Lareng, L. (Fac. Pharm. Biol. Sci., Rene Descartes Univ., Paris, France). *C. R. Acad. Sci.* [D] *(Paris)* 279:1565-1569, 1974.

1810 SHORT-TERM INHALATION TOXICITY OF HALOGENATED HYDROCARBONS. EFFECTS ON FASTING RATS. *(E.)* Jaeger, R. J.; Conolly, R. B.; Murphy, S. D. (Harvard Sch. Public Health, Boston, Mass.). *Arch Environ. Health* 30(1):26-36, 1975.

1811 ULTRASTRUCTURAL PATHOLOGY OF THE LIVER AFTER CHRONIC EXPOSURE TO HALOTHANE. *(E.)* Chang, L. W.; Dudley, Jr., A. W.; Katz, J. (Univ. Wisconsin Med. Sch., Madison). *Am. J. Pathol.* 78(1): 14a-15a, 1975.

1812 ULTRASTRUCTURAL ALTERATIONS IN RABBIT LIVER FOLLOWING ENDOTOXIN ADMINISTRATION. *(E.)* Tanksley, D. R.; Lockard, V. C.; Brunson, J. G. (Univ. Mississippi Med. Cent., Jackson). *Am. J. Pathol.* 78(1):11a-12a, 1975.

1813 HEPATIC ANGIOSARCOMA IN MAN AND RODENTS FOLLOWING PROLONGED EXPOSURE TO VINYL CHLORIDE. *(E.)* Gordon, D. E.; Thomas, L. B.; Kent, G.; Calandra, J.; Bahu, R.; Popper, H. (Ind. Biotest Inc., Northbrook, Illinois). *Gastroenterology* 67(4):794, 1974.

1814 CHANGES IN THE PARAMETERS OF MITOCHONDRIAL RESPIRATION AND THE CONTENTS OF SOME NUCLEOTIDES IN THE RAT HEPATOCYTE AFTER IN VIVO CONTAMINATION WITH PLUTONIUM (^{230}Pu) CITRATE. *(Fr.)* Pepin, G.; Pasquier, C.; Vallee, C.; Duprey, F.; Boudene, C. (Lab. Toxicol., Univ. Paris-Sud, Chatenay-Malabry, France). *C. R. Acad. Sci.* [D] *(Paris)* 280:141-144, 1975.

1815 IN VIVO STABILITY OF PLUTONIUM (^{239}Pu) AS THE CITRATE. STUDY OF ITS EXCRETION AND HEPATIC UPTAKE IN THE RAT. *(Fr.)* Boudene, C.; Pepin, G. (Lab. Toxicol., INSERM, Univ. Paris-Sud, France). *C. R. Acad. Sci.* [D] *(Paris)* 279:1677-1680, 1974.

1816 THE EFFECT OF PHENOBARBITONE ON PROTEIN SYNTHESIS BY LIVER POLYRIBOSOMES IN FED AND STARVED RATS. *(E.)* Ragnotti, G.; Aletti, M. G. (Cent. Cell. Pathol., Univ. Milan, Italy). *Biochem. J.* 146(1):1-12, 1975.

1817 LACK OF INFLUENCE OF THE MEMBRANES OF THE ENDOPLASMIC RETICULUM ON THE CAPACITY FOR PROTEIN SYNTHESIS OF THE RAT LIVER POLYRIBOSOMES. *(E.)* Ragnotti, G.; Aletti, M. G.; Cipollini, T. L. (Cent. Cell. Pathol., Univ. Milan, Italy). *Biochem. J.* 146(1):13-15, 1975.

1818 REGULATION OF ORNITHINE DECARBOXYLASE ACTIVITY BY PUTRESCINE AND SPERMIDINE IN RAT LIVER. *(E.)* Janne, J.; Holtta, E. (Dep. Med. Chem., Univ. Helsinki, Finland). *Biochem. Biophys. Res. Commun.* 61(2):399-406, 1974.

1819 THE ROLE OF THE GLYCEROLPHOSPHATE SHUTTLE IN HETEROGENOUS LIVER MITOCHONDRIA. *(E.)* Lamartiniere, C. A.; Weiss, G. (Dep. Obstet. Gynecol., Columbia Univ., N.Y.). *Hoppe Seylers Z. Physiol. Chem.* 355(12):1549-1554, 1974.

1820 EFFECTS OF COPPER-LOADING IN RABBITS ON THE SUBCELLULAR DISTRIBUTION OF LIVER COPPER, ACID PHOSPHATASE, ACID β-GLUCURONIDASE AND NEUTRAL THIAMINE PYROPHOSPHATASE: SOME COMPARISONS WITH COPPER-LOADING IN RATS. *(E.)* Campbell, Jr., W. G. (Emory Univ. Sch. Med., Atlanta, Ga.). *Am. J. Pathol.* 78(1):12a-13a, 1975.

1821 STUDIES ON THE INDUCTION AND REPRESSION OF ENZYMES IN RAT LIVER. CHARACTERISTICS OF THE L-TRYPTOPHAN AND CORTISONE-MEDIATED INDUCTION OF SERINE DEHYDRATASE IN THE LIVERS OF INTACT AND ADRENALECTOMIZED RATS. *(E.)* Cihak, A.; Inoue, H.; Pitot, H. C. (Univ. Wisconsin Med. Sch., Madison). *Arch. Biochem. Biophys.* 166(1):237-244, 1975.

1822 THE MODIFYING EFFECT OF MANGANESE ON THE ENZYMIC PROFILES AND PATHWAYS OF CARBOHYDRATE METABOLISM IN RAT LIVER AND ADIPOSE TISSUE DURING DEVELOPMENT. *(E.)* Baquer, N. Z.; Hothersall, J. S.; Greenbaum, A. L.; McLean, P. (Middlesex Hosp. Med. Sch., London, England). *Biochem. Biophys. Res. Commun.* 62(3):634-641, 1975.

1823 HEPATOCELLULAR UPTAKE OF FERRITIN IN THE RAT. *(E.)* Unger, A.; Hershko, Ch. (Hadassah Univ. Hosp., Jerusalem, Israel). *Br. J. Haematol.* 28(2):169-179, 1974.

1824 OPTICAL MEASUREMENT OF THE CATALASE-HYDROGEN PEROXIDE INTERMEDIATE (COMPOUND I) IN THE LIVER OF ANESTHETIZED RATS AND ITS IMPLICATION TO HYDROGEN PEROXIDE PRODUCTION IN SITU. *(E.)* Oshino, N.; Jamieson, D.; Sugano, T.; Chance, B. (Sch. Med., Univ. Pennsylvania, Philadelphia). *J. Biochem.* 146(1):67-77, 1975.

1825 STIMULATION OF THE PATHWAY OF PORPHYRIN SYNTHESIS IN THE LIVER OF RATS AND MICE BY GRISEOFULVIN, 3,5-DIETHOXYCARBONYL-1,4-DIHYDROCOLLIDINE AND RELATED DRUGS: EVIDENCE FOR TWO BASICALLY DIFFERENT MECHANISMS. *(E.)* De Matteis, F.; Gibbs, A. H. (Med. Res. Counc. Lab., Carshalton, England). *Biochem. J.* 146(1):285-287, 1975.

1826 SEXUAL DIFFERENCES IN HEPATIC METABOLISM AND INTRACELLULAR DISTRIBUTION OF CORTICOSTERONE STUDIED BY PULSE LABELING WITH (1,2,6,7-^{3}H) CORTICOSTERONE. *(E.)* Carlstedt-Duke, J.; Gustafsson, J. A.; Gustafsson, S. A. (Dep. Chem., Karolinska Inst., Stockholm Sweden). *Biochemistry* 14(3):639-648, 1975.

1827 EFFECT OF FASTING ON THE CONTENT OF ORGANIC FREE RADICALS PRESENT IN RAT LIVER. *(Fr.)* Mosora, F.; Marechal, R.; Duchesne, J. (Dep. At. Mol. Phys., Univ. Liege, Belgium). *C. R. Acad. Sci. [D] (Paris)* 279:1557-1559, 1974.

1828 THE STRUCTURE OF BILIRUBIN AND BILIVERDIN IN COMPLEXES WITH SERUM ALBUMIN. *(Ger.)* Blauer, G.; Wagniere, G. (Dep. Biol. Chem., Heb. Univ., Jerusalem, Israel). *Chimia* 29(1):20, 1975.

1829 ENZYMATIC DETERMINATION OF BILE ACIDS IN STOOL SPECIMENS. *(Ger.)* Reimold, W. V.; Kattermann, R. (Med. Clin., Univ. Gottingen, Germany). *Z. Gastroenterol.* 12(5):341-348, 1974.

1830 BILIARY EXCRETION OF SULFONAMIDE. I. BILIARY EXCRETION OF SULFADIMETHOXINE. *(Jpn.)* Miyazaki, K.; Ogino, O.; Kamiya, A.; Hori, R.; Arita, T. (Hokkaido Univ. Hosp., Japan) *J. Pharm. Soc. Jpn.* 94(9):1090-1094, 1974.

1831 EFFECT OF ADMINISTRATION OF A COLONIC METABOLITE OF CHOLIC ACID ON CHOLESTEROL LEVELS IN BILE AND BLOOD. *(E.)* Pomare, E. W.; Low-Beer, T. S. (Bristol R. Infirm., England). *Gut* 15(10):830, 1974.

See also, 1603, 1612, 1613, 1652, 1683, 1688, 1841, 1851, 1871, 2202, 2206, 2348.

1832 ENZYME HISTOCHEMICAL STUDY OF FAT ABSORPTION IN HUMAN DUODENAL MUCOSA. *(E.)* Monges, H.; Chamlian, A.; Cougard, A.; Mathieu, B. (North Hosp., Marseille, France). *Gut* 15(10):777-782, 1974.

In 7 healthy male subjects (30-62 yr) the activities of 10 intestinal enzymes were studied using a fasting control peroral biopsy and 2 other biopsies 15 and 30 min after an intestinal infusion of emulsified corn oil. The long chain fatty acids in the corn oil were distributed as 9% palmitic acid, 3% stearic acid, 46% oleic acid, 41% linoleic acid, and 1% arachidic acid. Four healthy subjects with a nutritional status identical to the tested subjects did not receive any fat and were submitted to 3 biopsies at 15-min intervals to detect if any enzymatic modification was induced by the biopsy tube. Histochemical methods were used to compare the enzymatic activities of the absorptive cells at the tips of the villi before and during fat absorption which was demonstrated with a Sudan black stain. Four of the 10 oxidative enzymes were modified by the corn oil infusion: NADH tetrazolium reductase, NADPH tetrazolium reductase, glucose-6-phosphate dehydrogenase (G-6-PD), and lactate dehydrogenase (LD). In 6 cases, the activity of NADH tetrazolium reductase was increased. Two of these subjects presented a simultaneous increase of NADPH tetrazolium reductase and G-6-PD; 2 other subjects presented a decrease in LD activity. These histochemical data indicate that after corn oil has been instilled into the lumen, conspicuous, enzymatic modifications occur in the absorptive cells at the tips of the duodenal villi when the fat absorption is important.

1833 ALKALINE PHOSPHATASES IN THE SMALL-INTESTINAL WALL OF THE RAT AFTER JEJUNO-ILEAL TRANSPOSITION. *(E.)* Gronqvist, B.; Lundqvist, G. (Univ. Hosp., Uppsala, Sweden). *Acta. Chir. Scand.* 140(8): 643-648, 1974.

To further elucidate the ability of jejunum and ileum to retain their functions after transposition, the activity of alkaline phosphatases in the wall of the small intestine was studied. Fasted Sprague-Dawley rats were subjected to surgical jejuno-ileal transposition (transposed group); controls were sham-operated or left unoperated. Analyses of alkaline phosphatases in homogenates of intestinal tissue were performed 2, 6, and 12 weeks after surgery. Compared with the controls, the transposed animals showed a somewhat reduced weight gain. There was no appreciable difference in protein content/ml homogenate between controls and transposed rats. The activity of alkaline phosphatases in the intestinal segments studied contradicted the hypothesis that the enzyme activity of the transposed segments adapts completely to their new positions. The results indicate that with the exception of certain initial enzyme losses, each intestinal segment essentially retains its original enzyme activity after transposition and that the intestinal cells cannot be stimulated to above-normal enzyme activity by nutrition from small bowel contents.

1834 THE EFFECT OF DIFFERENT DIETS ON THE ALKALINE PHOSPHATASE, ADENOSINE TRIPHOSPHATASE (ATPase) AND DISACCHARIDASE LEVELS OF THE SMALL INTESTINE OF THE RAT. *(E.)* Hietanen, E. (Dep. Physiol., Univ. Turku, Finland). *Comp. Biochem. Physiol.* 50(1A):41-46, 1975.

The effects of conventional diets on the mucosal enzyme levels in the small intestine was studied using 4 groups of male Wistar rats. Pelleted diets from 3 different manufacturers were used; the pellets fed to groups I and II originated from the same manufacturer, but those fed to group II contained more vitamins, proteins, and fats. The data showed marked differences in the mucosal alkaline phosphatase, ATPase, and disaccharidase activities among the 4 groups after feeding on various pellets for 4 weeks. No significant changes had been detected after 2 weeks feeding. Groups I and II had very marked differences in the enzyme activities at 4 weeks. The weight gain and liver:body weight ratio, and hepatic and intestinal protein contents showed only minor variations, indicating that the dietary requirements of the rats were satisfied by each of the pelleted diets. The lactase activity varied most markedly in the small intestine of rats on different pelleted diets. Even maltase and trehalase activities had nearly significant differences from the control group after alteration of the pelleted diet. These data emphasize the fact that even minor changes in the content of pelleted diets and diets from various commercial manufacturers may induce marked changes in the mucosal enzyme levels. This might be one reason for the frequently discrepant results on mucosal enzyme levels found when comparing data from various laboratories. It is suggested that the source of the diet and its content should be noted, as even the slightest variations may be reflected in the mucosal metabolism. These results also support an earlier study which indicated that the adaptation of mucosal enzymes to a diet was not detectable at 2 weeks but was detectable after 4 weeks time.

1835 THE ACTIVITY OF INTESTINAL DISACCHARIDASES AND DIPEPTIDASES IN RATS WITH CHRONIC RENAL INSUFFICIENCY. *(E.)* Grimmel, K.; Bongartz, S.; Kasper, H. (Med. Univ., Wurzburg, Germany). *Acta Hepatogastroenterol. (Stuttg.).* 21(5):383-387, 1974.

The activity of intestinal disaccharidases and dipeptidases was studied in 18 male Wistar rats with chronic renal insufficiency (induced by 5/6-nephrectomy and subsequent resection with thermocautery). A sham operation was given to 19 controls. Homogenates from small intestinal mucosa were used to estimate the activities of the disaccharidases (lactase, cellobiase, saccharase and maltase) and the dipeptidases which hydrolyse the dipeptides L-leucyl-L-leucine, L-leucyl-L-proline, L-leucyl-glycine, glycyl-L-leucine, L-methionyl-L-leucine, L-methionyl-L-methionine and L-methionyl-L-proline. Only the mean saccharase activity was decreased in the nephrectomized uremic rats (65.4 IU/g protein) as compared to that in the controls (93.2 IU/g protein). The mean activity for

the dipeptidases hydrolyzing L-leucyl-L-proline and L-methionyl-L-proline was significantly increased in the nephrectomized rats. Also the increase in activity of L-leucyl-L-proline dipeptidase correlated positively with severity of uremia (as indicated by increased blood urea-nitrogen values). Thus the authors have found a significant decrease in the saccharase activity of uremic rats and, in contrast to previous reports, no significant changes in the activities of lactase, cellobiase and maltase; on the other hand they have found significant increases in 2 dipeptidases.

1836 MULTIPLE FORMS OF PEPTIDE HYDROLASES FROM GUINEA-PIG INTESTINAL MUCOSA. *(E.)* Piggott, C. O.; Fottrell, P. F. (Dep. Biochem., Univ. College, Galway, Ireland). *Biochem. Soc. Trans.* 2(6):1366-1368, 1974.

Beta peptide hydrolases from guinea pig mucosal homogenates were purified and characterized. For purification of β_1 and β_2 hydrolases, mucosal scrapings were homogenized in sodium borate buffer (pH 8.4) and the supernatant centrifuged. The enzyme activity was precipitated with solid $(NH_4)_2SO_4$ in borate buffer (pH 8.4) and dialyzed against this buffer. The dialyzed solution was applied to a DEAE cellulose column and chromatographed by a linear salt-gradient elution procedure. Fractions containing enzyme activity were pooled, concentrated and subjected to preparative polyacrylamide-gel electrophoresis. The β_1 and β_2 hydrolases had pH optima at pH 8.2 (which is different from that for α peptide hydrolase). Varying degrees of inhibition of the enzymes were observed with Co^{++}, Mn^{++}, Cu^{++} and Zn^{++}. There was no endopeptidase activity as measured with denatured cytochrome C and no activity was found with N-benzyloxycarbonly-Gly-L-Phe. The enzymes hydrolyzed a wide range of peptides and many of the peptide substrates were hydrolyzed by both. They were particularly active with glycine-containing dipeptides (in contrast with α peptide hydrolase). Although the β_1 and β_2 peptide hydrolases differed in their affinities for various peptides and had slightly different electrophoretic mobilities other similarities indicate they are closely related enzymes.

1837 ACID α-GLUCOSIDASE FROM HUMAN GASTROINTESTINAL MUCOSA -- SEPARATION AND CHARACTERIZATION. *(E.)* Asp, N. G.; Gudmand-Hoyer, E.; Christiansen, P. M.; Dahlqvist, A. (Chem. Cent., Univ. Lund, Sweden). *Scand. J. Clin. Lab. Invest.* 33(3):239-245, 1974.

The separation and characterization of α-glucosidase, an intestinal maltase with an acid pH-optimum, is described and the extent that this enzyme contributes to the total maltase activity in crude mucosal homogenates from various parts of the human gastrointestinal tract at various pH was determined. Homogenates of mucosa from the stomach (corpus and antrum), the duodenum and the jejunum were used. The enzyme was separated using gel filtration chromatography on a Bio-Gel P-300 column. Maltase activity in the stomach was higher when measured at pH 4 than at pH 6. Also, maltase activity in the duodenum was optimum at pH 4-4.5, but in the jejunum it was optimum at pH 5.5-6. In homogenates from the stomach nearly all the fractions with maltase activity were soluble at both pH 4 and 6. In duodenal homogenates the major fraction of the activity was soluble only when measured at pH 4, and in jejunal homogenates fractions with most of the activity were insoluble. The activity of the separated acid α-glucosidase was maximal at pH 4.5 as tested on various substrates (isomaltose, maltose, p-nitrophenyl α-glucoside and amylose). The K_m values with the latter 3 substrates were approximately 10^{-2} M/liter, 3.5×10^{-3} M/liter and 5 mg/ml, resp., regardless of the source of enzyme. Maltose was hydrolyzed more rapidly than any of the other substrates tested. Turanose inhibited the enzyme activity, while sucrose did not. Thus this investigation has demonstrated that there is a soluble acid α-glucosidase in the human gastrointestinal mucosa that appears to be responsible for almost all of the maltase activity in normal gastric mucosa but contributes just a small percent to the overall maltase when the jejunum is included.

1838 AMINO ACIDS IN GUT CONTENTS AND BLOOD PLASMA OF RATS AS AFFECTED BY DIETARY AMINO ACID IMBALANCE. *(E.)* Nasset, E. S.; Ju, J. S. (Dep. Nutr. Sci., Univ. California, Berkeley). *J. Nutr.* 105(1):69-79, 1975.

The effect of an amino acid imbalanced diet on amino acid homeostasis in blood plasma and gut contents in male Sprague-Dawley rats was studied. Six series of 13 rats were fed a stock diet and after 6 days blood and gut contents were obtained from one rat in each series and analyzed for amino acids. The remaining 12 rats in each series were divided into 2 equal groups and fed a control and an imbalanced diet. Both diets contained equal concentrations of casein (5%) that on complete hydrolysis would yield 48 g of amino acids/kg of diet. The imbalanced diet was produced by adding 56 g of essential amino acids to each kg of diet. Blood and gut contents were obtained from these rats on each day of the 6 day test and chromatographic columns were used for amino acid analysis. Ingestion of the imbalanced diet resulted in a rise in total plasma amino acids, while the initial response to the control diet was an immediate drop of total amino acids followed by a gradual rise. The change in total plasma amino acids for both diets was relatively small, but there were large differences among individual amino acids, some of which changed in opposite directions. Threonine exhibited the most striking changes: the gut content values rose to over 200% of the stock diet values and the plasma values increased to 320% at day 6 for the imbalanced diet. It was suggested that dietary amino acid imbalance affects amino acid homeostasis in ways not yet understood, but that certain unspecified amino acid interactions may be operative.

1839 INTESTINAL CALCIUM-BINDING PROTEIN IN ANIMALS FED NORMAL AND RACHITOGENIC DIETS: II. PIG STUDIES. *(E.)* Harrison, J. E.; Hitchman, A. J. W.; Brown, R. G. (Dep. Med., Univ. Toronto, Canada). *Can. J. Physiol. Pharmacol.* 53(1):144-149, 1975.

The relationship between intestinal concentrations of calcium-binding protein and the presence of rickets was studied in pigs. For 6 weeks before breeding, throughout gestation, and throughout the 4-week nursing period, a sow was maintained on a standard diet minus vitamin D; the sow was kept in an area free of ultraviolet light. Six of the weanling pigs were placed for 5 months on the same vitamin D-deficient diet; half the litter was given a single i.m. dose of vitamin D_2 (500,000 IU) 3 days before slaughter. The duodenal mucosa from all pigs was examined for calcium-binding protein and the results compared with those from control pigs fed the standard diet with usual vitamin-D supplements. In a 2nd experiment, weanling pigs from a sow fed a normal diet were maintained for 12 weeks on a vitamin D-deficient, calcium-deficient, low-phosphate diet; 1/2 the litter was injected i.m. with vitamin D_2 (1,000,000 IU) 72 hr before slaughter. In the 1st experiment, the pigs fed the vitamin D-deficient diet showed neither biochemical nor radiological evidence of rickets. Calcium-binding protein was present in the duodenal mucosa but to a lesser extent than in the controls; vitamin D_2 injection before death increased the calcium-binding protein concentration by a factor of 2. In the 2nd experiment, the pigs fed the low-phosphate, calcium-deficient, vitamin D-deficient diet were underweight and showed clinical and histological signs of rickets. Serum calcium and serum phosphorus values were below normal, but calcium-binding protein was present in normal concentrations in the duodenal mucosa. Calcium-binding protein and serum calcium and phosphorus levels were unaffected by vitamin D_2 injection before death. The vitamin D levels in the livers of the rachitic pigs were less than 1/20 those in control animals. The data indicate that intestinal calcium-binding protein concentrations cannot be used as a reliable indication of the presence or absence of rickets.

1840 ACTIVE SECRETION OF CALCIUM, SODIUM AND CHLORIDE BY ADULT RAT DUODENUM IN VITRO. *(E.)* Walling, M. W.; Kimberg, D. V. (Harvard Med. Sch., Boston, Mass.). *Biochim. Biophys. Acta* 382(3): 213-217, 1975.

Active Ca^{2+} secretion across proximal rat duodenum was studied at Ca^{2+} concentrations which should saturate unidirectional mucosal to serosal flux but not serosal to mucosal flux. Ion fluxes were studied *in vitro* with a modified Ussing apparatus using duodenums from adult male Holtzmann rats. The tissues were perfused with media containing 25 mM $CaCl_2$; control media contained similar concentrations of Cl^- and were of similar osmolarity. Calcium and Na^+ fluxes were measured simultaneously using $^{45}Ca^{2+}$ and $^{22}Na^+$ as tracers. While there was no net Ca^{2+} flux across the duodenum when the medium Ca^{2+} concentration was 1.25 mM, there was a large active secretion of Ca^{2+} when the level was raised to 25 mM. With the latter concentration, unidirectional mucosal to serosal flux was increased only by a factor of 10, while serosal to mucosal flux increased in proportion to the change in Ca^{2+} concentration. Active Na^+ secretion was of similar magnitude at both Ca^{2+} concentrations. Both unidirectional Na^+ fluxes and intestinal conductance were decreased at 25 mM Ca^{2+}. Cellular metabolism and some intracellular transport processes were apparently unaffected. When Na^+, Ca^{2+}, and Cl^- fluxes were measured simultaneously across stripped rat duodenum under conditions with no co-transportable substrate (hexose or amino acid) in the mucosal solution, active secretion of Cl^- and Na^+ was observed, along with a small absorption of Ca^{2+}. The sum of Na^+ and Cl^- fluxes did not account for the entire short-circuit current measured. Bicarbonate ion secretion may account for the residual ion flux. The results support the hypothesis that the polarity of net Na^+ and Cl^- fluxes changes from secretion to absorption between the proximal and distal small bowel.

1841 A STUDY OF THE CYTOPLASMIC RECEPTORS FOR GLUCOCORTICOIDS IN INTESTINE OF PRE- AND POSTWEANLING RATS. *(E.)* Henning, S. J.; Ballard, P. L.; Kretchmer, N. (Dep. Pediatr., Stanford Univ., Calif.). *J. Biol. Chem.* 250(6):2073-2079, 1975.

To determine the mechanism of glucocorticoid-induced enzymic and morphologic changes in the rat intestine during weaning, the cytoplasmic fraction of 18-day-old rat intestines was examined for specific glucocorticoid-binding proteins. Incubation of intestinal slices from Wistar rats with (^{3}H)dexamethasone in a physiological medium at 2 C showed the presence of a cytoplasmic binding macromolecule with high specificity for steroids having glucocorticoid activity. The binding reaction was saturable and of high affinity. Binding was reversible on addition of nonlabeled dexamethasone, indicating that the usual assay procedure measured both corticosterone-filled and unoccupied binding sites. Sucrose density gradient centrifugation indicated that the receptor-dexamethasone complex from intestinal cytosol sedimented at the same rate as that from liver (8.2 sedimentation coefficient). The receptor-dexamethasone complex was stable at 2 C for at least 24 hr in intestinal slices, but in isolated cytosol fractions there was a considerable loss of binding even in the presence of high concentrations of (^{3}H)dexamethasone. The presence of cytosol from intestinal mucosa, but not that from muscle layers, caused a dissociation of dexamethasone from liver cytosol receptors, suggesting the presence of some interfering factor in isolated mucosal cytosol. The instability of steroid-receptor complexes in the presence of isolated intestinal cytosol seems to be associated with homogenization, thus having no physiological significance. Receptor activity was present in the intestine from late fetal life through adulthood, but the concentrations were significantly higher during the first 2 weeks after birth than at all other times. In contrast, receptor activity in rat lung cytosol was high at the time of birth, while that in the liver rose steadily during the 1st postnatal week and remained at high levels thereafter. Thus, specific receptors for glucocorticoids are present in the rat intestine during periods of responsiveness and unresponsiveness. This suggests that although corticosteroids exert their effects through the cytoplasmic receptors, this early event in glucocorticoid action may not be a controlling step for changes in responsiveness during development.

1842 CELLULAR LOCALIZATION OF A VASOACTIVE INTESTINAL PEPTIDE IN THE MAMMALIAN AND AVIAN GASTROINTESTINAL TRACT. *(E.)* Polak, J. M.; Pearse, A. G. E.; Garaud, J. C.; Bloom, S. R. (R. Postgrad. Med. Sch., Hammersmith Hosp., London, England). *Gut* 15(9):720-724, 1974.

A series of immunofluorescence-based studies investigated the localization of vasoactive intestinal polypeptide in the mammalian (2 mongrel dogs, 1 pig, and 1 baboon) and avian (30 Japanese quail) gastrointestinal mucosa. An antiserum to a pure porcine vasoactive intestinal polypeptide, possessing no cross reactivity against the related hormones glucagon, secretin, and gastrin-inhibitory peptide, revealed a wide distribution of vasoactive intestinal polypeptide cells throughout the entire length of the mammalian and avian gut. In the mammalian gastrointestinal tract, satisfactory results were obtained only in the freeze-dried and diethylpyrocarbonate vapor-fixed preparations incubated in the presence of trasylol. The highest numbers of vasoactive intestinal polypeptide cells were found in the large intestine of all 3 mammalian species and also in the body of the stomach in both the pig and baboon. In the avian gastrointestinal tract, good results were obtained with all 3 preparative techniques: (1) methanol-free formaldehyde, paraffin embedding; 2) 10% carbodiimide, cryostat sections; and 3) diethylpyrocarbonate, but these preparations were always superior. Vasoactive intestinal polypeptide cells were present in all regions except the gizzard. There were only 2 endocrine cell types that could be identified by electron microscopy in the avian gut and these were correlated with the argentaffin and argyrophil varieties on light microscopy. Sequential immunofluorescence and silver staining, showed that all the vasoactive intestinal polypeptide cells in the avian gastrointestinal tract belonged to the argyrophil variety. On electron microscopy, 3 types of endocrine cells in the mammalian gut were sufficiently widely distributed to be considered as the sites for production of vasoactive intestinal polypeptide: 1) enterochromaffin cells; 2) enteroglucagon cells; and 3) D cells. Serial section immunofluorescence for enteroglucagon and vasoactive intestinal polypeptide in mammalian gut samples indicated that the 2 cells were not identical and that each was differently localized in the mucosa. The finding that the avian vasoactive intestinal polypeptide cell is not the argentaffin enterochromaffin cell can perhaps be extrapolated to mammalian gut. These results suggest the D cell of the Wiesbaden classification as the most likely site for the production of vasoactive intestinal polypeptide. The final identification cannot be made, however, until a suitable preparative technique which allows the peptide to be demonstrated immunologically in resin-embedded semi-thin sections has been developed.

1843 THE AUTONOMIC CONTROL OF COLONIC MUCIN SECRETION IN THE MOUSE. *(E.)* Smith, B.; Butler, M. (St. Bartholomew's Hosp., London, England). *Br. J. Exp. Pathol.* 55(6):615-621, 1974.

The autonomic control of the synthesis and secretion of colonic mucin was studied in male Porton mice. Some animals received drugs p.o. and others i.p. in a wide range of doses in both short- and long-term experiments. Carbachol was used as a parasympathetic stimulator, atropine as a parasympathetic blocker, norepinephrine as an α-adrenergic stimulator, guanethidine as an α-adrenergic blocker, isoprenaline as a β-adrenergic stimulator, and propanolol as a β-adrenergic blocker. All tissues were fixed for 24 hr in 10% formol saline and blocks taken from the proximal and distal colon. Paraffin sections were stained with periodic acid/Schiff, alcian blue and high iron diamine to show the degree of acidity and sulfation of the mucins. The amount of sulfomucin was greatest in the distal colon and rectum and decreased proximally towards the cecum. There was an unbroken layer of sialomucin covering the epithelium of the lumen and the crypts. It could be seen in the lumen both loose and surrounding any foreign material there with a thick layer. Sulfomucin also was released in large masses to lie in the lumen or against the wall. A sialomucin protected the mucosa so that even full thickness trauma initiated no inflammatory response. Carbachol produced considerable epithelial shedding, resulting eventually in large mucosal deficiencies, but the mucin layer remained intact and there was no ulceration or inflammatory reaction. β-Adrenergic stimulating drugs produced increased mucin synthesis and secretion, swelling of epithelial cells and inflammatory infiltration of the mucosa with retention of cellular debris and polymorphs in the crypts. β-Adrenergic blocking agents produced hemorrhage and inflammation acutely, and in the long-term, shortening and thickening of the muscle coats probably due to ischemia. These findings suggest that the β-adrenergic system has more important effects on the structure and function of colonic mucosa than the cholinergic system.

1844 ACIDIFICATION IN THE RAT PROXIMAL JEJUNUM. *(E.)* Blair, J. A.; Lucas, M. L.; Matty, A. J. (Dep. Chem., Univ. Aston, Birmingham, England). *J. Physiol. (Lond.)* 245(2):333-350, 1975.

The cause of acidification in the rat proximal jejunum was investigated using the everted sac technique to measure the production of H^+. Jejunal sacs were taken from fasted male Wistar rats. Acidification was measured in phosphate buffer in closed and open systems; in some cases, the incubation flasks were left to stand in ice water before or after incubation. Acidification occurred in the absence of glucose, but increased on the addition of glucose to reach maximal values. An apparent K_m of 1.78 mM was derived for the glucose-dependent process. Acidification in the presence of glucose was inhibited by 10 mM of 2,4-dinitrophenol, phlorrhizin, aminophylline, or anaerobiosis. Histamine, EDTA, ouabain, and acetazolamide, compounds which are known to alter acid production in the gastric mucosa, had no effect on jejunal acidification. The unmetabolizable sugars, galactose and 3-O-methylglucose, also failed to increase acidification, while the metabolizable sugars mannose and fructose did lead to increases. Serosal and mucosal lactate production were measured and the calculated percentage of hydrogen ions possibly derived from

this source was shown to account for only a small percentage of the acidification. The greatest increase in acidification with minimal simultaneous production of lactate occurred on the addition of ATP to the incubation medium, which did not enter the intestinal tissues. The data indicate that acidification is caused by hydrolysis at the mucosal surface of ATP derived from intracellular metabolic sources. The acid microclimate postulated to exist at the surface of the jejunal mucosa could be created by an aqueous phase at the mucosal surface not in equilibrium with the bulk phase, a source of hydrogen ions at the mucosal surface and slow diffusion of such ions within this phase. A microclimate with replenishment of hydrogen ions from jejunal surface acidification could affect the rate of transfer of weak acids and should be reconsidered as a way in which weakly ionizing substances are transferred.

1845 THE IN VIVO METABOLISM OF CHOLESTEROL BY GUT BACTERIA IN THE RAT AND GUINEA-PIG. *(E.)* Goddard, P.; Hill, M. J. (Cent. Public Health Lab., London, England). *J. Steroid Biochem.* 5(7): 569-572, 1974.

The metabolism and excretion of [C_4-^{14}C]cholesterol was studied in 7 Sprague-Dawley rats and 5 guinea pigs. Cholesterol was injected into the proximal cecum of anesthetized animals under direct surgical vision, the incision was closed, and urine and feces were collected for 72 hr and chromatographically and spectrophotometrically analyzed for radioactive metabolites. About 88% of the total radioactivity was recovered in the fecal neutral steroid fraction of rats and guinea pigs; only 0.3 and 1.7%, resp., was recovered from urine. Analysis of the neutral steroid fraction showed that the major metabolite was 5β-cholestan-3β-ol (coprostanol), accounting for 57.3% and 69% of the total radioactivity in rats and guinea pigs, resp. Rat and guinea pig feces also contained small amounts of 4-cholesten-3-one (5.7% and 4.3%, resp.); rat but not guinea pig feces also contained 5β-cholestan-3-one (capostanone, 2.0%). Unmetabolized cholesterol comprised 35 and 26.7% of the fecal neutral steroid fraction in rats and guinea pigs, resp. Acid steroids (lithocholic and isolithocholic acid) made up 6.3% of the rat fecal cholesterol metabolites but were not excreted by guinea pigs. ^{14}C-estradiol and estrone, identified in the urine of guinea pigs, were reduced 5-fold by pretreatment of animals with antibiotics. Negligible amounts of ^{14}C were recovered in blood and bile and about 0.5% of the total dose was found in guinea pigs 24 hr after cholesterol instillation. Since previous studies have shown that cholesterol is poorly absorbed from the large bowel and only a small fraction of serum cholesterol is converted to urinary estrogens or to acid steroids, it was concluded that cholesterol is metabolized by the gut flora. The cholesterol side chain is completely removed in the guinea pig whereas only a 3-carbon unit is removed in the rat.

1846 COMPARISON OF SECRETORY AND HISTOLOGICAL EFFECTS OF SHIGELLA AND CHOLERA ENTEROTOXINS IN RABBIT JEJUNUM. *(E.)* Steinberg, S. E.; Banwell, J. G.; Yardley, J. H.; Keusch, G. T.; Hendrix, T. R. (John Hopkins Univ. Sch. Med., Baltimore, Md.). *Gastroenterology* 68(2):309-317, 1975.

The secretory responses to maximal doses of shigella toxin or cholera toxin were studied to determine if mucosal damage is a prerequisite for toxin-induced secretion. Two 15-20 cm segments of proximal jejunum from male New Zealand rabbits (2-3 kg) were isolated and cannulated. The toxin solutions contained either shigella enterotoxin in concentrations ranging from 10-650 μg/ml or cholera enterotoxin in a concentration of 0.5 g of a freeze dried culture preparation mixed with 40 ml of distilled water. Shigella toxin levels of 200 μg/ml and above resulted in consistent secretion, but a concentration above 200 μg/ml did not further increase secretion. Significant fluid production occurred only after 105 min exposure. The maximum rate of secretion produced by shigella toxin was identical to that observed with cholera toxin. The secretory effect of the toxins were not additive. Epithelial alterations were observed in postperfusion biopsies, but the changes were not significant compared with controls, suggesting an effect from perfusion rather than from the toxins. Only minimal inflammation was observed. It is concluded that both shigella and cholera toxin induce secretion in the absence of histological changes.

1847 ADENYLATE CYCLASE IN INTESTINAL CRYPT AND VILLUS CELLS: STIMULATION BY CHOLERA ENTEROTOXIN AND PROSTAGLANDIN E_1. *(E.)* Schwartz, C. J.; Kimberg, D. V.; Ware, P. (Harvard Med. Sch., Boston, Mass.). *Gastroenterology* 68(1):94-104, 1975.

The adenylate cyclase activities of villus and crypt epithelial cells were compared under basal conditions and after stimulation by fluoride, cholera enterotoxin, and prostaglandin E_1 (PGE_1). Mucosal fractions were prepared from the ileum of New Zealand male white rabbits with a planning technique and from the distal small intestine of albino male Sherman rats by a vibration technique. Incubations were initiated by adding 20 μl of homogenate containing approximately 200 μg of protein to 30 μl of medium. The final reaction mixture consisted of 50 mM Tris-HCl buffer at pH 7.5, 5 mM $MgCl_2$, 10 mM caffeine, 0.2mM ATP, 1 μCi of [8-^{14}C]ATP, and an ATP-regenerating system consisting of 10 mM phosphoenolypyruvate and 12.5 μg of pyruvate kinase. Incubations were performed for 5 min at 37 C. Just prior to termination of the reaction, 0.2 ml of a solution containing 60 μg of cyclic AMP and 0.25 μCi of cyclic [^{3}H]AMP was added to each tube. The incubation medium was then passed over a 0.5 x 3.0 cm column containing Dowex 50 W-X4 resin. Basal activity, as well as that stimulated by 10 mM NaF, was assessed in fractions from both control and cholera enterotoxin-exposed rabbit loops. Basal, NaF, and PGE_1-stimulated activities were measured in fractions obtained from rat intestine. In both species, base line adenylate cyclase activity was greater in crypt than in villus cells. After exposure to cholera enterotoxin *in vivo*, adenylate cyclase activity was enhanced 2-7 times in all fractions prepared from rabbit ileum, and the response was most marked in villus cells. Adenylate

cyclase in membranes prepared from both rat villus and crypt intestinal cells was consistently doubled over the base line activity by the *in vitro* addition of PGE_1; NaF led to a 5- to 7-fold increase. These results indicate that both villus and crypt cells contain one of the important components required for the cyclic AMP-mediated secretory response, namely, a cholera enterotoxin and PGE_1-sensitivity adenylate cyclase activity. Since an increased level of cyclic AMP alone may not be sufficient to evoke a secretory response, it is concluded that these studies do not clarify the extent to which each of these major cell types may participate in cyclic AMP-mediated secretion.

1848 THE RESPONSE OF SMALL INTESTINAL VILLOUS AND CRYPT EPITHELIUM TO CHOLERATOXIN IN RAT AND GUINEA PIG. EVIDENCE AGAINST A SPECIFIC ROLE OF THE CRYPT CELLS IN CHOLERAGEN-INDUCED SECRETION. *(E.)* De Jonge, H. R. (Dep. Biochem., Erasmus Univ., Rotterdam, Netherlands). *Biochim. Biophys. Acta* 381(1):128-143, 1975.

Kinetic properties of adenylate cyclase in separately isolated upper villous and crypt cells from rat and guinea pig small intestine were compared to determine the relative contribution of villous and crypt epithelium to the choleragen-induced secretion *in vivo*. An apparent K_m of 0.4 mM was found for both enzymes in the rat. The V-values measured in the fluoride-stimulated state indicated an approximately equal enzyme content of both cell populations as opposed to a preferential localization in the brush border region of the epithelial cell. Adenylate cyclase was irreversibly activated in both villous and crypt compartments by prolonged contact of the small intestine with luminally administered choleragen. The maximal stimulation of the upper villous enzyme (4-7 times) exceeded the maximal effect on the crypt enzyme by 2-fold. A lag phase of at least 30 min was noted between the first luminal contact with the purified choleragen and a significant activation of the adenylate cyclase associated with isolated intestinal brush borders from the upper villous region. Adenylate cyclase activity in the upper villus could be optimally stimulated in the absence of any alteration of crypt cell activity by using a short exposure time (2 min) of the luminal surface to high amounts of choleragen. By comparing, *in vivo*, the effects of short and prolonged contact with cholera toxin on the unidirectional and net flux of ions and water in ileal and jejunal segments, it was concluded that both villous and crypt regions contribute to the secretion of water and electrolytes (Na^+, Cl^-, and HCO_3^-) during cholera. The serosal to mucosal flux of Na^+ and Cl^- increased without a significant alteration of the opposite flux. These results indicate that absorptive and secretory processes occur within the same epithelial compartment. The results of this study support the view that the crypt epithelium fulfills a specific role during the choleragen-induced secretion of ions and water.

1849 INTESTINAL METABOLISM OF PHENACETIN IN THE RAT: EFFECT OF CHARCOAL-BROILED BEEF AND RAT CHOW. *(E.)* Pantuck, E. J.; Hsiao, K. C.; Kuntzman, R.; Conney, A. H. (Coll. Physicians Surg., Columbia Univ., New York, N.Y.). *Science* 187:744-746, 1975.

The effects of charcoal-broiled ground beef and of rat chow diet on the metabolism of phenacetin were studied in the intestine of male Long-Evans rats. The proximal 5 cm segment of the small intestine was used to determine phenacetin O-dealkylation. The amount of *N*-acetyl-*p*-aminophenol (µg/5 cm intestine/90 min) in the tissue plus incubation solution represented the metabolism of phenacetin. A semisynthetic diet resulted in a mean value of 1.35; ground beef (cooked on foil), 1.97; ground beef (raw), 2.77; rat chow, 5.99; ground beef (charcoal-broiled), 15.88. The results indicate that charcoal broiled beef and a lab chow diet stimulate the intestinal metabolism of phenacetin in the rat. It is suggested that there is a need for studies in man to determine whether charcoal-broiled meat and other dietary constituents can stimulate the intestinal metabolism of phenacetin or other drugs and thereby decrease their absorption and bioavailability.

1850 EFFECTS OF LACTULOSE AND OTHER LAXATIVES ON ILEAL AND COLONIC pH AS MEASURED BY A RADIOTELEMETRY DEVICE. *(E.)* Bown, R. L.; Gibson, J. A.; Sladen, G. E.; Hicks, B.; Dawson, A. M. (St. Bartholomew's Hosp., London, England). *Gut* 15(12): 999-1004, 1974.

The effect of lactulose on luminal pH in the ileum and colon of normal subjects and patients without gastrointestinal disease was compared with that of 2 other laxative agents, magnesium sulphate and sodium sulphate. A pH-sensitive radiotelemetry capsule was swallowed by each subject and luminal pH values were recorded every hr after giving the laxative. The agents and dose ranges were: lactulose syrup (80-110 M/day), magnesium sulphate mixture (22-23 M magnesium salt/day), sodium sulphate solution (25-37.5 M day), and a mixture of 10 ml lactulose and 15 ml sodium sulphate solution (3 times/day). Lactulose induced a highly significant fall in pH in the right colon, and magnesium sulphate induced a significantly elevated rectal pH; also, pH values were significantly higher along the gastrointestinal tract in the magnesium sulphate group. Sodium sulphate significantly lowered the pH in the left colon and rectum compared with controls, but only in the rectum compared with those given lactulose. The mixture of lactulose and sodium sulphate lowered the pH in the right colon and rectum compared with controls, but showed no significant difference when compared to the pH values induced by lactulose alone. Thus, lactulose produced consistent and marked acidification of proximal colonic contents, but changes in the rest of the colon were less consistent. If acidification of proximal colonic contents is considered beneficial in patients with chronic hepatic encephalopathy, these studies suggest that lactulose would be more effective than sodium sulphate, and that the combination of the 2 laxatives is unlikely to be more effective than lactulose alone.

1851 RADIOIMMUNOASSAY OF CHOLECYSTOKININ-PANCREOZYMIN. *(E.)* Harvey, R. F.; Dowsett,

L.; Hartog, M.; Read, A. E. (Bristol R. Infirm., England). *Gut* 15(9):690-699, 1974.

A sensitive and specific radioimmunoassay was developed for cholecystokinin-pancreozymin (CCK-PZ) using rabbit antisera to crude porcine hormone. Highly purified porcine CCK-PZ, labeled with ^{131}I, and repurified by column chromatography on Sephadex G15, was used as tracer. Separation of free from antibody-bound labeled CCK-PZ was carried out using charcoal, ion-exchange resin, or a double antibody procedure. Non-specific interference with the assay system by serum factors was abolished by boiling and diluting the serum samples before assay. Ninety-nine % pure porcine CCK-PZ (standard), commercial CCK-PZ preparations, cerulein, the C-terminal 8- and 12-amino acid fragments of the CCK-PZ molecule, and endogenous human CCK-PZ all cross reacted in the assay system and showed parallel inhibition curves. No significant cross reaction was found with gastrin, secretin, glucagon, or insulin. The sensitivity of the assay is approximately 5 pg/ml of test solution, which proved adequate for measuring physiological levels of CCK-PZ in peripheral blood in man. Serum samples were obtained from 30 healthy volunteers (20-40 yr-old) after an overnight fast, and from 20 patients (40-75 yr-old) without upper gastrointestinal disorders. The mean immunoreactive CCK-PZ concentration in these 50 subjects was 60.4 pg/ml. The distribution of individual values was skewed, however, so that the median was much lower (30 pg/ml). Older subjects had higher fasting levels of CCK-PZ than the young adults. The lack of suitably characterized reference standards for either porcine or human CCK-PZ makes it difficult to compare the results of different assays or to determine with certainty the levels of endogenous human CCK-PZ. Even so, if properly selected control groups are used, it is suggested that the measurement of differences in CCK-PZ during physiological studies should be perfectly acceptable until well-characterized and stable standard preparations become available.

1852 STIMULATORY EFFECT OF A BRUSSELS SPROUTS OR CABBAGE DIET ON INTESTINAL DRUG METABOLISM IN THE RAT. *(E.)* Hsiao, K. C.; Pantuck, E. J.; Wattenberg, L. W.; Kuntzman, R.; Jacobson, M.; Conney, A. H. (Coll. Physicians Surg., Columbia Univ., New York, N.Y.). *Fed. Proc.* 34(3):742, 1975.

1853 LACK OF DIETARY ADAPTATION OF INTESTINAL 2-MONOGLYCERIDE ACYL TRANSFERASE. *(E.)* Rao, G. A.; Abraham, S. (VA Hosp., Martinez, Calif.). *Lipids* 9(11):940-942, 1974.

1854 FOOD-INDUCED CHANGES IN A DUODENAL FACTOR CONTROLLING THE INSULIN RESPONSE. *(Fr.)* Zermatten, A.; Felber, J. P. (Canton. Hosp., Univ. Lausanne, Switzerland). *Experientia* 30(6):648, 1974.

1855 THE EFFECT OF RAW SOYA BEAN ON IN VITRO ACTIVE AND PASSIVE ACCUMULATION BY RAT SMALL INTESTINE. *(E.)* Pope, H. O.; Patten, J. R.; Lawrence, A. L. (Univ. Texas Dent. Branch, Houston). *Br. J. Nutr.* 33(2):117-125, 1975.

1856 CALORIC INTAKE AND WEIGHT REGULATION IN WEANLING RATS WITH DORSOMEDIAL HYPOTHALAMIC LESIONS (DMNL). *(E.)* Bernardis, L. L.; Border, J. R. (Dep. Surg., State Univ. New York, Buffalo). *Fed. Proc.* 34(3):935, 1975.

1857 CONTROL OF FOOD INTAKE IN LEAN AND OBESE PIGLETS. *(E.)* Wangsness, P. J.; Gobble, J. L. (no affil.). *Fed. Proc.* 34(3):935, 1975.

1858 OBSERVATIONS OF PLASMA SECRETIN AND GASTRIN RESPONSES TO A MEAT MEAL AND DUODENAL ACIDIFICATION IN DOGS. *(E.)* Lee, K. Y.; Tai, H. H.; Chey, W. Y. (Genesee Hosp., Rochester, N.Y.). *Fed. Proc.* 34(3):441, 1975.

1859 INTERACTION OF DIETARY FAT AND INOSITOL ON PHOSPHOLIPID AND PHOSPHATIDYLINOSITOL METABILISM IN THE GUT OF THE GERBIL. *(E.)* Woods, M. N. (Harvard Sch. Public Health, Boston, Mass.). *Fed. Proc.* 34(3):932, 1975.

1860 POSTNATAL CHANGES IN LACTASE ACTIVITY IN THE INTESTINE OF TWO BREEDS OF PIGS. *(E.)* Ekstrom, K. E.; Grummer, R. H. (no affil.). *Fed. Proc.* 34(3):935, 1975.

1861 THE MECHANISMS OF ACID DISPOSAL BY RABBIT DUODENAL MUCOSA. *(E.)* Fiddian-Green, R. G.; Silen, W. (no affil.). *S. Afr. J. Surg.* 12(2):80, 1974.

1862 EFFECT OF LYSOLECITHIN ON IN VITRO BINDING OF RIBOSOMES TO MEMBRANES OF ROUGH ENDOPLASMIC RETICULUM (RER) OF RAT INTESTINAL MUCOSA. *(E.)* Yousef, I. M.; Kuksis, A. (no affil.). *Fed. Proc.* 34(3):916, 1975.

1863 CHOLECYSTOKININ ACTIVITY IN THE RAT SMALL INTESTINE. *(E.)* Schneeman, B. O. (Bruce Lyon Res. Lab., Oakland, Calif.). *Fed. Proc.* 34(3): 454, 1975.

1864 STUDIES ON THE NATURE OF CHOLECYSTOKININ-PANCREOZYMIN IN SMALL-INTESTINE MUCOSAL EXTRACTS. *(E.)* Harvey, R. F.; Dowsett, L.; Read, A. E. (Briston R. Infirm., England). *Gut* 15(10): 838-839, 1974.

1865 THE INFLUENCE OF ETHACRYNIC ACID AND ASPIRIN ON WATER AND ELECTROLYTE SECRETION INDUCED BY PROSTAGLANDIN E_1 IN THE HUMAN JEJUNUM. *(E.)* Matuchansky, C.; Mary, J. Y.; Rambaud, J. C.; Bernier, J. J. (INSERM, St. Lazare Hosp., Paris, France). *Gut* 15(10):831, 1974.

1866 THE CONTENT OF VITAMIN A FRACTIONS AND RETINYL-PALMITATE HYDROLASE ACTIVITY IN THE SMALL INTESTINE MUCOSA AND ITS CELL MEMBRANES IN VITAMIN A DEFICIENCY AND RADIATION EXPOSURE. *(Rus.)* Leutskii, K. M.; Liubovich, E. N.; Sovtysik, D. D. (Chernovtsy State Univ., USSR). *Dokl. Akad. Nauk SSSR* 220(4):972-974, 1975.

1867 NUCLEAR 1α,25-DIHYDROXYVITAMIN D_3 RECEPTORS IN INTESTINE. *(E.)* Brumbaugh, P. F.; Haussler, M. P. (Arizona Med. Cent., Tucson). *Fed. Proc.* 34(3):887, 1975.

1868 COMPARISON OF HISTOCHEMICAL AND DISC ELECTROPHORESIS FINDINGS ON POSTNATAL DIFFERENTIATION OF ENZYMES IN THE INTESTINAL MUCOSA. *(Ger.)* Hohn, P.; Schafer, A.; Paasch, W.; Gabbert, H. (Pathol. Inst., Univ. Mainz, Germany). *Verh. Dtsch. Ges. Pathol.* 57:444, 1973.

1869 THE PRODUCTION OF LYSOZYME BY THE PANETH CELL. *(E.)* Vantrappen, G. R.; Peters, T. L. (no affil.). *Gut* 15(10):826-827, 1974.

1870 MORPHOLOGICAL FINDINGS ON THE ROLE OF GRANULE-CONTAINING PANETH CELLS IN INTESTINAL CALCIUM SECRETION. *(Ger.)* Schafer, H. J.; Otto, H. F. (Pathol. Inst., Univ. Hamburg, Germany). *Verh. Dtsch. Ges. Pathol.* 57:445, 1973.

1871 KINETICS OF CELL PROLIFERATION IN PARENCHYMAL ORGANS AND IN THE INTESTINAL EPITHELIUM OF JUVENILE RATS. *(Ger.)* Stocker, E. (Pathol. Inst., Univ. Wurzburg, Germany). *Verh. Dtsch. Ges. Pathol.* 57:388, 1973.

1872 COMPLETE TRANSPLANTATION OF THE SMALL INTESTINE IN THE DOG: II. TWO COMPARATIVE SERIES OF ORTHOTOPIC AUTOGRAFTS AND HOMOGRAFTS. *(Fr.)* Vignal, J.; Tissot, E.; Moulay, A.; Papathanassiou, Z.; Faure, J. L. (Edouard Herriot Hosp., Lyon, France). *Lyon Chir.* 70(5):310-311, 1974.

See also, 1599, 1715, 1749.

GENERAL

1873 STABILITY OF NORMAL HUMAN FECAL FLORA DURING A CHEMICALLY DEFINED, LOW RESIDUE LIQUID DIET. *(E.)* Bornside, G. H.; Cohn, Jr., I. (Dep. Surg., Lousiana State Univ., New Orleans). *Ann. Surg.* 181(1):58-60, 1975.

The effect of feeding of a chemically defined, low residue diet (1200-1800 cal/day for 7 days) on fecal flora was studied in 10 healthy male volunteers. All stools were collected and 3 were cultured for aerobic and anaerobic bacteria: 1) a pre-diet specimen; 2) 1 on the 7th day of the diet; 3) one 3 days after resumption of a regular diet. During the low residue diet, total daily fecal output was decreased 70% with each subject passing only 2 or 3 stools over the 7-day period. Mean total aerobe and anaerobe counts were 10^7/g and 10^{10}/g, resp., throughout study. There was no overgrowth by opportunistic bacteria or fungi. Fecal flora were not altered nor was there any quantitative change in any bacterial group during the diet. It is concluded that the large numbers of bacteria remaining in the feces after a low residue diet do not warrant the sole use of such a regimen for preoperative colon preparation.

1874 ROLE OF INTESTINAL MICROBES ON BODY NITROGEN ACCUMULATION IN GERMFREE, GNOTOBIOTIC AND CONVENTIONAL MICE. *(E.)* Yamanaka, M.; Nomura, T.; Kametaka, M. (Cent. Inst. Exp. Anim., Tokyo, Japan). *J. Nutr. Sci. Vitaminol. (Tokyo)* 20(5):389-400, 1974.

The effects of intestinal microbes on the digestion and absorption of nutrients and on the retention of nitrogen were studied using 80-day-old germfree, *Escherichia coli* or *Staphylococcus epidermidis* monocontaminated (at 56 days of age) gnotobiotic, and conventional male mice. The semi-micro Kjeldahl method was used to measure total N and protein N in the carcasses and gut contents of these animals. The total body weight and liver and kidney weights were greater in the conventional mice than in the germfree or gnotobiotic mice. The total N/100 g body weight of the carcass was also greater among the conventional mice than among the germfree or gnotobiotic mice; the carcasses of the *S. epidermidis*-infected mice contained more N than those of the *E. coli*-infected mice, which in turn contained more total N than those of the germfree mice. There were no major differences among these groups in the accumulation of ^{15}N by the carcass, liver, or leg muscles 2 days after dietary administration of 0.2% ^{15}N-labeled secondary ammonium phosphate; however, the conventional mice tended to accumulate more ^{15}N than the other groups. The total N/100 g body weight in the intestinal contents was highest in the germ-free mice, followed by the *E. coli*-infected, *S. epidermidis*-infected, and conventional animals. Nitrogen levels in the cecal contents of the germ-free and gnotobiotic mice were considerably higher than those in the cecal contents of the conventional mice. The ratio of protein N:total N in the cecum, colon, and rectum was significantly higher in the conventional animals than in the others, and the ratio was lower in both gnotobiotic groups than in the germfree animals. The ratio throughout the rest

of the gut was basically the same in all 4 groups, and the groups did not differ in total Cr_2O_3/U body weight in the stomach, colon, and rectum. However, the cecal and small intestinal Cr_2O_3 contents of the conventional mice were lower than those of the gnotobiotic and germfree animals. Except in the colon and rectum, total N/100 mg Cr_2O_3 was higher in the gut contents of the conventional mice than in the other mice. The results indicate that intestinal microbes affect the nutrition of the host, *S. epidermidis* alone having a beneficial effect.

1875 CHARACTERIZATION OF HUMAN COLONIC MUCOPROTEIN ANTIGEN. *(E.)* Gold, D.; Miller, F. (Health Sci. Cent., State Univ. New York, Stony Brook). *Immunochemistry* 11(7):369-375, 1974.

Human colonic mucoprotein antigen was purified and its physical, chemical and immunological characteristics are described. The crude antigen preparation was extracted using a phenol-water solvent system. Concentrations of 0.1-10.0 mg/ml of the antigen were used. Descending paper chromatography was used for the qualitative determination of monosaccharides, and amino acid analysis and hexosamine ratios were determined by using a Beckman Amino Acid Analyzer Model 120C. A Hewlett-Packard Model 402 Gas Chromatograph was used for the determination of individual neuraminic acids. The purified antigen was found to be a sulfated mucoprotein with N-glycolylneuraminic acid, galactosamine, glucosamine, galactose and fucose being the only monosaccharides present (in the molar ratios of 19:23:23:26:10, resp.). The antigen was also found to be rich in threonine, serine, proline, glycine and glutamic acid. The molecular weight was estimated to be 1.5×10^7 daltons. Colonic mucoprotein antigen appears chemically similar to blood group substance A. However there was no immunological cross reactivity between the 2 molecules, nor was there an immunological similarity between colonic mucoprotein antigen and carcinoembryonic antigen as determined by double diffusion and immunoelectrophoresis. It is suggested that this colonic mucoprotein antigen may be endogenous in ulcerative colitis and may be a specific antigen for the detection of colonic carcinoma.

1876 COMPARISON OF METABOLIC ACTIVITY OF RUMEN MICROFLORA IN LAMBS AND ADULT SHEEP: IN VITRO UTILIZATION OF UREA NITROGEN AND SUGARS. *(Fr.)* Barry, J. L.; Durand, M. (Lab. Technol. Anim. Food, Jouy-en-Josas, France). *C. R. Acad. Sci.* [*D*] *(Paris)* 279:1373-1376, 1974.

1877 EXPERIMENTAL AND CLINICAL EXPERIENCE IN SUBCUTANEOUS SPLENIC TRANSPOSITION TO PREVENT BLEEDING FROM OESOPHAGEAL VARICES. *(E.)* Bengmark, S. (no affil.). *S. Afr. J. Surg.* 12(2): 64, 1974.

1878 IS THERE AN ANTRAL-BODY PORTAL SYSTEM IN THE STOMACH? *(E.)* Taylor, T. V.; Torrance, H. B. (R. Infirm., Manchester, England). *Gut* 15(10):836, 1974.

1879 THE TRANSILLUMINATION TECHNIQUE IN STUDYING INTRAMURAL BLOOD FLOW TO ASSESS THE VIABILITY OF THE GASTRIC WALL. *(Rus.)* Druzhkov, B. K. (V. I. Lenin Inst. Postgrad. Med., Kazan, USSR). *Eksp. Khir. Anesteziol.* (6):31-33, 1974.

1880 THE CIRCULATION OF THE SMALL BOWEL MUCOSA. *(E.)* Lundgren, O. (Dep. Physiol., Univ. Goteborg, Sweden). *Gut* 15(12):1005-1013, 1974.

1881 GEOPHAGIA AND MINERAL NUTRITION IN WILD PRIMATES. *(Fr.)* Hladik, C. M.; Gueguen, L. (Mus. Nat. Hist., Brunoy, France). *C. R. Acad. Sci.* [*D*] *(Paris)* 279:1393-1396, 1974.

1882 RENAL INACTIVATION OF SECRETIN AND CHOLECYSTOKININ. *(Ger.)* Lehnert, P.; Stahlheber, H.; Forell, M. M.; Fullner, R.; Fruhauf, S. (2nd Med. Clin., Univ. Munich, Germany). *Z. Gastroenterol.* 12(4):269-272, 1974.

1883 THE EXISTENCE OF A SPECIAL APPETITE-REGULATING HORMONE, ARENTERIN, IN THE INTESTINE. *(Rus.)* Skvortsova, N. N.; Volkonskaia, V. A.; Tuliaganova, E. Kh.; Zabolotnykh, V. A.; Ugolev, A. M.; Khokhlov, A. S. (I. P. Pavlov Inst. Physiol., Leningrad, USSR). *Dokl. Akad. Nauk SSSR* 220(2):493-495, 1975.

1884 SURFACE AREA AND EFFECT OF DRYING TEMPERATURE RELATED TO DRY WEIGHT OF JEJUNAL TISSUE IN RAT. *(E.)* Lucas, M. L.; Johnson, I. T. (Dep. Physiol., Ludwig-Maximillian Univ., Munich, Germany). *Experientia* 30(12):1487-1488, 1974.

See also, 1815.

DIAGNOSTIC PROCEDURES

1885 ANGIOGRAPHY IN ACUTE INTESTINAL ISCHEMIA. *(Fr.)* Poilleux, J.; Bigot, R.; Monnier, J. P.; Hivet, M. (St. Antoine Hosp., Paris, France). *Ann. Chir.* 27(6):623-625, 1973.

Intestinal ischemia can be caused by 3 conditions: (1) aortic dissection; (2) emboli, usually in the superior mesenteric artery; and (3) thrombosis resulting from acute arteritis. All of these lesions can be diagnosed by angiography. Surgical possibilities in (1) are limited to those rare patients who can undergo prosthetic replacement of the aorta and reimplantation of the splanchnic and renal blood vessels. In (2), if angiography shows the embolism is located in the proximal superior mesenteric artery, a limited intestinal resection may be performed. If the embolism is located in the distal superior mesenteric artery, medical therapy is indicated. If the patient fails to respond or if chronic ischemia of the small intestine develops 2-3 months later, a segmental resection of the small intestine should be performed. Thrombosis can be diagnosed only by lateral aortography. If the superior mesenteric artery is thrombosed, as is most often the case, treatment consists of reimplantation associated with more or less extensive intestinal resection. In some cases, thrombosis of the gastrointestinal arteries may necessitate double celiac and superior mesenteric revascularization. For the diagnosis of acute intestinal ischemia it is recommended that lateral and ventral aortography always be performed and, if necessary, selective arteriography.

1886 SELECTIVE PHLEBOGRAPHY OF THE PANCREAS. *(E.)* Gothlin, J.; Lunderquist, A.; Tylen, U. (Univ. Hosp., Lund, Sweden). *Acta Radiol.* 15(5): 474-480, 1975.

A new method is presented for selective catheterization of the pancreatic veins by a transhepatic approach. Of 40 patients, 7 were examined by this method, 4 with carcinoma of the bile ducts with no pancreatic involvement, 2 with pancreatic carcinoma, and 1 with portal hypertension. The catheter was inserted by the usual technique for transhepatic cholangiography, guided into the main stem of the portal vein, and then into the pancreatic veins as in the transumbilical technique. The remaining 33 patients were controls with a malignant tumor outside the pancreatico-duodenal area, on whom the transumbilical approach was used. The pancreatic veins had an abnormal appearance in the 2 patients with pancreatic carcinoma. In 1 several venous branches were obstructed and others were irregularly invaded. In the other the posterior superior pancreatico-duodenal vein was compressed, but catheterization was still possible. There were no major complications in the controls, but 2 of the patients given the transhepatic technique developed intraabdominal bleeding. These 2 patients had jaundice, however, giving them additional risk of bleeding. It is concluded that pancreatic phlebography may be a technique of some clinical importance in the diagnosis of pancreatic lesions.

1887 CHANGES IN THE SERUM LIPASE ACTIVITY AFTER ENDOSCOPIC RETROGRADE PANCREATOGRAPHY. *(Ger.)* Bornschein, W.; Weiss, H. D.; Kramann, B. (2nd Med. Clin., Munich Tech. Univ., Germany). *Dtsch. Med. Wochenschr.* 99(41):2028-2032, 1974.

Serum lipase activities were measured before and immediately after retrograde endoscopic pancreatography and 24 and 48 hr thereafter in 46 randomly selected patients. Before endoscopy, 13 patients had increased lipase activities: 11 of these 13 had radiological evidence of changes in the pancreatic ducts (chronic pancreatitis, pancreatic abscess associated with chronic pancreatitis, pancreatic neoplasms, and dysontogenetic cysts). The remaining 2 patients had acute and chronic hepatitis, resp., with normal pancreatic ducts. Although none of these patients developed clinical symptoms of pancreatitis, 36 of the 46 patients had serum lipase levels as high as 6380 U/liter/min immediately after endoscopy. In all cases, these lipase activities decreased significantly after 24 hr and were in the normal range in 20 of the 36 patients after 48 hr. The remaining 16 patients consisted of the 13 with initially elevated lipase activities and 3 patients who had unusually large increases after endoscopy. A correlation was found between the increase in serum lipase levels and the extent to which the pancreatic ducts were filled. Because of the small number of cases, this correlation was significant only for patients with filling of the main and accessory ducts and for those in whom the parenchyma was visualized. The average rate at which serum lipase levels decreased was independent of changes in the pancreatic duct, parenchymal visualization, and the initial level of lipase activity.

1888 PHOSPHATASES. VII. ALKALINE PHOSPHATASE IN BIOPSY SPECIMENS OF NORMAL AND DAMAGED HUMAN LIVER. *(E.)* Stepan, J.; Volek, V.; Balas, V.; Vecerek, B. (Fac. Med., Charles Univ., Prague, Czechoslovakia). *Acta Hepatogastroenterol. (Stuttg.)* 21 (6):425-429, 1974.

The isozymes of liver alkaline phosphatase were studied in liver biopsy specimens from patients with normal and non-cholestatically diseased livers, and compared with those in the serum. Controlled or blind needle biopsies were taken from the right and left hepatic lobes from 6 patients with fatty liver disease, eight with cirrhosis, 10 with chronic hepatitis, and nine with non-cholestatic gallbladder disease. Nine patients without liver disease served as controls. Total liver alkaline phosphatase activity was determined for 10 mM 4-nitrophenylphosphate and referred to g wet tissue weight and mg protein. The remainder of liver homogenate was subjected to electrophoresis for 60 min at 3 C, 30 V/cm and 5 mA/cm. In all patients, serum liver alkaline phosphatase isozymes were determined by inactivation-inhibition and agar gel electrophoresis. In the liver tissue, only the chronic hepatitis patients showed a significant increase in liver alkaline phosphatase activity. In the serum, the total activity and liver isozyme of serum liver alkaline phosphatase were significantly increased in all patients with diseased livers. Gel electrophoresis showed 3 bands of enzyme activity in liver tissue. Slow anodic was found in normal and gallbladder disease patients. Fast anodic was found in most of the fatty liver cases

and 70% of the chronic hepatitis cases. A cathodic band was found in 1/3 of the cirrhotic patients. Electrophoretically identical bands were found in the serum. It is concluded that there is no direct diagnostic effect of liver alkaline phosphatase patterns in liver tissue, but that they do correlate with serum phosphatase patterns.

1889 SINGLE-VISIT ORAL CHOLECYSTOGRAPHY. *(E.)* Burhenne, H. J.; Obata, W. G. (Child. Hosp., Adult Med. Cent., San Francisco, Calif.). *N. Engl. J. Med.* 292(12):627-628, 1975.

Fractionated 2 day administration of 6 g iopanoic acid followed by a single radiography session on a 3rd day was studied on 611 ambulatory patients undergoing cholecystography. Each patient was put on a low fat diet for 2 days during which 12 tablets were taken; 4 on day 1 and 8 on day 2. A single X-ray examination was made and classified as adequate visualization, poor visualization, or non-visualization. Fifty patients had adequate visualization of a normal gallbladder; 31, adequate visualization of cholelithiasis; and 1 of cholesterol polyps. Twenty had non-visualization, 7 due to extrinsic causes, 11 had operative confirmation of cholelithiasis; and 2 were not followed up. Six patients had poor visualization due to diarrhea but had adequate visualization with the calcium salts of sodium ipodate. The findings correlate statistically with those patients given 1 day administration of iopanoic acid. This fractionation technique eliminates the need for repeat oral cholecystography, often found with iopanoic acid administration.

1890 ENDOSCOPIC RETROGRADE CHOLANGIOGRAPHY (ERC) IN PATIENTS WITH JAUNDICE. *(E.)* Classen, M.; Koch, H.; Schwamberger, K.; Demling, L. (Med. Clin., Univ. Erlangen, Germany). *Rend. Gastroenterol.* 6(2):116-120, 1974.

The value of endoscopic retrograde cholangiography in the differential diagnosis of jaundice was investigated, and the results compared with those obtained by puncture techniques. Of 114 patients (all with serum bilirubin values over 4.0 mg%), positive findings were obtained by endoscopic retrograde cholangiography in 85 cases (74.5%). These included: choledocholithiasis (40); exclusion of extrahepatic jaundice (15); carcinoma of the head of the pancreas (seven); tubular stenosis of the common bile duct (five); common bile duct stenosis and papillary stenosis without demonstrated stones (four each); contracted choledochoduodenostomy with food in the bile duct system (three); papillary injury and tumor of the efferent bile ducts (two each); and intrahepatic concrements, cholelithiasis and choledocholithiasis with perforation of stone into the colon, and primary sclerosing cholangitis (one each). The cause of jaundice was also determined by duodenoscopy and endoscopic retrograde cholangiography in an additional seven patients, bringing the total of positively-diagnosed cases to 80.7%. The use of endoscopic retrograde cholangiography did not succeed in visualization of the efferent bile ducts in the remaining 22 cases. Because puncture techniques such as percutaneous transhepatic cholangiography and laparoscopic cholecystocholangiography are contraindicated in the presence of hemorrhage; are reported to be successful in only about 50% of cases; and have complication rates of up to 5% (the transhepatic technique), it is concluded that endoscopic retrograde cholangiography should be first considered for diagnosis in cases of jaundice.

1891 ESOPHAGUS, STOMACH, AND DUODENUM: RADIOLOGICAL DIAGNOSIS. *(Ger.)* Fernholz, H. J. (Med. Fac., Rhenish-Westphal. Tech. Coll., Aachen, Germany). *Therapiewoche* 24(17):1865-1871, 1974.

1892 ESOPHAGUS, STOMACH, AND DUODENUM: FUNCTIONAL DIAGNOSIS. *(Ger.)* Hausamen, T. U.; Fritsch, W. P. (1st Med. Clin. A., Univ. Dusseldorf, Germany). *Therapiewoche* 24(17):1848-1860, 1974.

1893 CLINICAL APPLICATION OF MANOMETRIC STUDIES OF THE ESOPHAGUS. *(Sp.)* Nogueira, J. R. (Spanish Hosp., Mexico City). *Galicia Clin.* 45(9): 831-839, 1973.

1894 ESOPHAGUS AND STOMACH: ENDOSCOPIC DIAGNOSIS. *(Ger.)* Frotz, H. (Med. Clin., Univ. Cologne, Germany). *Therapiewoche* 24(17): 1872-1879, 1974.

1895 GASTRIC SCINTIGRAPHY IN THE DIAGNOSIS OF CANCER. *(Ger.)* Judin, L. A.; Kusnezov, N. A.; Selichova, V. V. (I. M. Sechenov 1st Moscow Med. Inst., USSR). *Radiol. Diagn. (Berl.)* 14(6): 758-759, 1973.

1896 THE DIAGNOSTIC VALUE OF CONTROLLED BIOPSIES OF GASTRIC TUMORS. *(Ger.)* Hampel, K. E.; Stopik, D.; Niedobitel, F. (Westend Clin., Free Univ., Berlin, Germany). *Z. Gastroenterol.* 12(5):315-320, 1974.

1897 CONTRIBUTION OF GASTROSCOPY TO THE STUDY OF GASTRIC CANCER: OUR EXPERIENCE. *(Sp.)* Herrerias, J. M.; Navarro, F.; Osorio, M.; Jurado, M.; Jimenez, J.; Aljama, P.; Damiano, A.; Chamorro, J. (Fac. Med., Cadiz, Spain). *Rev. Esp. Enferm. Apar. Dig.* 44(7):697-706, 1974.

1898 METOCLOPRAMIDE IN RADIOLOGICAL FUNCTIONAL DIAGNOSIS OF THE STOMACH AND INTESTINE. *(Ger.)* Schulz, H. G.; Gottschild, D. (Radiol. Clin., Karl Marx Univ., Leipzig, Germany). *Dtsch. Z. Verdau. Stoffwechselkr.* 33(6):299-307, 1973.

1899 INTESTINAL ENDOSCOPY: LITERATURE REVIEW. *(Rus.)* Malinovskii, N. N.; Starikova, V. B.; Fenomenov, A. M. (All-Union Res. Inst. Clin. Exp. Surg., Moscow, USSR). *Khirurgiia (Mosk.)* (11): 120-127, 1974.

1900 DUODENAL ENDOSCOPY WITH CATHETERIZATION OF THE PAPILLA. *(Ger.)* Franken, F. H.; Janssen, M.; Falck, J.; Voigt, K. (St. Josef Hosp., Wuppertal-Elberfeld, Germany). *Therapiewoche* 24(17): 1880-1886, 1974.

1901 THE VALUE OF OUTPATIENT FIBERSIGMOIDOSCOPY. *(Ger.)* Jenny, S.; Fumagalli, I.; Hugel, H.; Nuesch, H. J.; Deyhle, P. (Dep. Intern. Med., Univ. Zurich, Switzerland). *Z. Gastroenterol.* 12(1):46, 1974.

1902 OCCULT BLOOD IN THE STOOLS AS A SCREENING TEST FOR COLORECTAL CANCER AND PRECANCEROUS POLYPS: A FIELD STUDY OF 815 ADULTS. *(Ger.)* Gnauck, R. (German Diagn. Clin., Wiesbaden). *Z. Gastroenterol.* 12(4):239-250, 1974.

1903 THERMOGRAPHY IN ACUTE APPENDICITIS. *(Rus.)* Orlov, N. S.; Cherniaev, Iu. S. (Arkhangelsk Med. Inst., USSR). *Klin. Khir.* (8):5-11, 1974.

1904 NEW MATHEMATICAL TECHNIQUE FOR ELIMINATING THE LIVER FROM THE PANCREATIC SCINTIGRAM. *(Ger.)* Markwardt, J.; Nauber, G.; Rosche, G.; Oczko, B. (Cent. Inst. Cancer Res., Berlin-Buch, Germany). *Radiol. Diagn. (Berl.)* 14(6):757-758, 1973.

1905 A PANCREATIC FUNCTION TEST USING COMBINED, CONTINUOUS SECRETIN AND PANCREOZYMIN STIMULATION. *(Ger.)* Seiffert, U. B.; Roth, W. D. (Cent. Intern. Med., Johann Wolfgang Goethe Univ., Frankfurt/Main, Germany). *Z. Gastroenterol.* 12(2):87-94, 1974.

1906 SIGNIFICANCE OF A NEW METHOD FOR THE DIAGNOSIS OF PANCREATIC DISEASE. *(It.)* Scarabelli, L.; Bonandrini, L. (Inst. Clin. Gen. Surg. Surg. Ther., Univ. Pavia, Italy). *Arch. Ital. Chir.* 97(1):101-107, 1973.

1907 CLINICAL AND EXPERIMENTAL PRINCIPLES FOR THE ENZYME DIAGNOSIS OF ACUTE PANCREATITIS. *(Rus.)* Starodubtseva, L. N.; Bolgova, G. D. (Rostov Med. Inst., USSR). *Klin. Khir.* (9):30-35, 1974.

1908 THE DIAGNOSTIC VALUE OF ENDOSCOPIC PANCREATOGRAPHY (ERP) IN PANCREATITIS: CORRELATION WITH EXOCRINE FUNCTION. *(Ger.)* Ammann, R.; Deyhle, P.; Fumagalli, I.; Hofmann, K. (Dep. Intern. Med., Univ. Zurich, Switzerland). *Z. Gastroenterol.* 12(1):20-21, 1974.

1909 RETROGRADE CHOLANGIOPANCREATOGRAPHY: TECHNICAL PROBLEMS, COMPLICATIONS, AND VALUE OF THE TECHNIQUE. *(Ger.)* Witzel, L.; Halter, F.; Fluckiger, J.; Ludin, J.; Vogeli, E. (Inselspital, Bern, Switzerland). *Z. Gastroenterol.* 12(1):22-23, 1974.

1910 DIAGNOSIS OF HEPATOLENTICULAR DEGENERATION WITH A RADIOACTIVE COPPER TEST. *(Ger.)* Biesold, D.; Bunther, K.; Lobe, J.; Lossner, J. (Neurol. Clin., Karl Marx Univ., Leipzig, Germany). *Radiol. Diagn. (Berl.)* 14(6):760, 1973.

1911 ADVERSE REACTIONS TO BROMOPHTHALEIN WHEN USED TO TEST FOR LIVER FUNCTION: REVIEW OF THREE PATIENTS. *(Ser.)* Tomasevic, M.; Tadic-Tigerman, V.; Jankovic, J.; Aganovic, V. (Mil. Hosp., Sarajevo, Yugoslavia). *Vojnosanit. Pregl.* 31(5):336-338, 1974.

1912 AN APPROACH TO THE DIAGNOSIS OF LIVER DISORDERS USING ^{123}I-BSP. *(E.)* Reuben, A.; Narasimah, K.; Myers, M.; Wood, T.; Britton, K. (Middlesex Hosp., London, England). *Gut* 15(10):837, 1974.

1913 EXPERIMENTAL STUDY OF THE DETECTION OF DEFECTS IN HEPATIC SCINTIGRAMS BY USING A COLLIMATOR WITH HIGH RESOLUTION. *(Ger.)* Brase, A.; Jahns, E.; Ohlendorf, M. (Med. Coll., Hanover, Germany). *Strahlentherapie* 48(4):359-365, 1974.

1914 HEPATIC SCANNING IN THE DIAGNOSIS OF LIVER ABSCESS. *(E.)* Rizk, G. K.; Massabni, S. R.; Mudarris, F. (Amer. Univ. Med. Cent., Beirut, Lebanon). *Leban. Med. J.* 27(5):543-550, 1974.

1915 LIVER ASPIRATION CYTOLOGY. *(E.)* Brits, C. J. (no affil.). *S. Afr. Med. J.* 48 (53):2207-2214, 1974.

1916 PERCUTANEOUS NEEDLE BIOPSY OF THE LIVER AND LARGE BILE DUCT OBSTRUCTION. *(E.)* Morris, J. S.; Gallo, G. A.; Scheuer, P. J.; Sherlock, S. (R. Free Hosp., London, England). *Gut* 15(10):824, 1974.

1917 THE SITE OF INTERCOSTAL LIVER BIOPSY. *(E.)* Mikulecky, M.; Hantak, I. (1st Med. Clin., Comenius Univ., Bratislava, Czechoslovakia). *Acta Hepatogastroenterol. (Stuttg.)* 21(6):419-421, 1974.

1918 DIFFERENTIAL DIAGNOSIS OF CHRONIC LIVER DISEASE WITH SEROCHEMISTRY BY USING DISCRIMINANT ANALYSIS WITH SPECIAL CONSIDERATION OF THE MESENCHYMA. *(Ger.)* van Husen, N.; Eberhardt, G.; Gerlach, U.; Intorp, H. W.; Oberwittler, W. (Med. Clin., Univ. Munster, Germany). *Z. Gastroenterol.* 12(5):327-334, 1974.

1919 CAN HB Ag BE DETECTED IN LIVER BIOPSIES WITH THE LIGHT MICROSCOPE? COMPARISON OF HB Ag IN SERUM WITH RESULTS FROM ALDEHYDE-THIONINE STAINING. *(Ger.)* Vogel, H. M.; Henning, H.; Luders, C. J.; von Braun, H. H.; Tripatzis, I. (Fohrenkamp Clin., Molln/Lauenburg, Germany). *Z. Gastroenterol.* 12(4):257-264, 1974.

1920 THE CLINICAL IMPORTANCE OF DETERMINING VARIOUS IMMUNOGLOBULINS IN VIRAL HEPATITIS IN CHILDREN. *(Ukr.)* Trishkova, L. O. (Kiev Med. Inst., USSR). *Pediatr. Akush. Ginekol.* (5):7-8, 1974.

1921 DUODENOGRAPHIC RADIOLOGICAL DIAGNOSIS OF THE PAPILLA OF VATER. IV. THE DIFFERENTIAL DIAGNOSIS OF DISEASES OF THE PAPILLA. *(Ger.)* Kamieth, H. (no affil.). *Radiologe* 14(3):149-153, 1974.

1922 CYTOLOGICAL STUDIES OF THE DUODENAL CONTENTS IN THE DIAGNOSIS OF BILIARY DISEASES. *(Rus.)* Orlova, V. P.; Kapral, Kh. A. (Tallin Br. Hosp., Baltic Railroad, USSR). *Klin. Med.* 51(11): 84-88, 1973.

1923 SIGNIFICANCE OF LIVER FUNCTION STUDIES IN AMOEBIASIS. *(E.)* Menkurkar, P. R.; Merchant, H. C.; Rao, P. R.; Satoskar, R. S. (L. T. M. Med. Coll., Bombay, India). *J. Postgrad. Med.* 20(4):166-175, 1974.

See also, 1952, 2222, 2247, 2332, 2390.

GASTROINTESTINAL DISEASES

ESOPHAGUS

1924 BIOFEEDBACK CONTROL OF LOWER ESOPHAGEAL SPHINCTER CONTRACTION IN MAN. *(E.)* Schuster, M. M.; Nikoomanesh, P.; Wells, D. (Baltimore City Hosp., Md.). *Proc. Int. Symp. Gastrointest. Motility, 4th, 1973* pp. 139-144.

Studies were conducted on 3 normal subjects and 6 patients with gastroesophageal reflux symptoms to determine whether lower esophageal sphincter pressures could be controlled following operant conditioning. Sphincter pressure was monitored by manometer in the supine resting subject. All subjects, while observing the meter, were instructed to cause the meter reading to increase without changing respiration or using voluntary muscles. On the average, normal subjects increased lower esophageal sphincter pressure by 75% during training (maximum increase = 14 mm Hg) whereas no changes occurred in intraesophageal or gastric pressures. Patients with reflux increased sphincter pressure during conditioning by as much as 5-6 mm Hg. One reflux patient, however, showed a sharp drop in sphincter pressure (-6 mm Hg). These studies suggest that control of lower esophageal sphincter pressure is possible by operant techniques and further suggest that mediation by voluntary muscle is not necessary to accomplish this.

1925 RELATIONSHIP BETWEEN CHANGES IN PLASMA GASTRIN AND LOWER OESOPHAGEAL SPHINCTER PRESSURE AFTER MEALS. *(E.)* McCall, I. W.; Harvey, R. F.; Owenc, C. J.; Clendinnen, B. G. (R. Infirm., Bristol, England). *Br. J. Surg.* 62(1):15-18, 1975.

Lower esophageal sphincter pressures and plasma gastrin levels were measured and compared in 20 volunteers after a standard protein meal. Symptomatic eosphageal reflux was observed in 3 and the remaining 17 had normal gastrointestinal tracts. After a 6 hr fast, all had basal esophageal sphincter pressures measured, and venous blood samples were radioimmunoassayed for gastrin content. Subjects were given a standard meal of 8.0 g meat extract in 300 ml water at pH 7.0. Sphincter pressures and blood samples were taken at 5, 10, 15, 20, 30, 45, 60, and 75 min after the meal. There was a significant rise in gastrin levels from 27.3-74.4 pg/ml within 20 min in all but 3 subjects. There was also a significant rise in sphincter pressure of 53.4% after 43.0 min. In 5 subjects, gastrin levels did not change appreciably, and pressures rose slightly but not significantly (18.0%). These 5 included the 3 with gastroesophageal reflux. Correlation between the peak rises of gastrin level and sphincter pressure was not significant until the 2 subjects with the highest gastrin level rises were removed. It is suggested that low sphincter pressure and associated reflux may be due to a defect in gastrin release from the stomach.

1926 THE RESPONSE OF THE LOWER OESOPHAGEAL SPHINCTER TO MAXIMAL DOSES OF PENTAGASTRIN. *(E.)* Trindade, L. M.; Rosenberg, I. L.; Rozycki, Z. J.; Giles, G. R. (St. James's Hosp., Leeds, England). *Br. J. Surg.* 62(1):11-14, 1975.

Responses of the lower esophageal sphincter and of gastric secretion to pentagastrin were investigated in patients with dyspepsia but no gastroesophageal reflux and in patients with hiatus hernia and reflux. After a 12 hr fast, subjects swallowed a multiple tube system for pressure measurements and gastric aspiration. Gastric secretion was collected for 1 hr, after which sphincteric pressure was measured for a 5 min period. A 6 g/kg dose of pentagastrin was given i.m. and the lower esophageal sphincter pressure was measured at 0, 4, 8, 10, 15, 20, 30, 40, 50 and 60 min after injection. Gastric secretion was collected in 10 min aliquots between pressure measurements. All subjects showed an increase in sphincteric pressure and gastric acid output following injection. The 16 subjects with dyspepsia showed an increase in sphincteric pressure of 87.43%, and acid ouput rose from 4.64 mEq/hr to 39.3 mEq/hr. The 9 subjects with hiatus hernia and reflux showed a pressure increase of 110.2% and acid output rose from 5.4 to 36.0 mEq/hr. These values were not significantly different from the 1st group. Four subjects with hiatus hernia but no reflux showed a pressure increase of 85.8%. Acid output rose from 4.9 to 32.9 mEq/hr. No statistical comparison was possible with the other 2 sets. It is concluded that there is no statistically significant correlation between sphincter response to pentagastrin in dyspeptic and hiatus hernia patients, nor between sphincter pressure and acid output.

1927 OESOPHAGEAL MOTOR DYSFUNCTION IN PATIENTS WITH DIVERTICULA OF THE MID-THORACIC OESOPHAGUS. *(E.)* Kaye, M. D. (Univ. Colo. Med. Cent., Denver). *Thorax* 29(6):666-672, 1974.

Manometric studies were performed on 12 patients with diverticula of the mid-thoracic esophagus. In 8 patients the diverticulum was near the interbranchial level, in 2 between the middle and distal thirds, and in 2 others within the distal third. Patients were studied in a supine position after an overnight fast, by means of 3 polyvinyl catheters. Lower esophageal sphincter contractions were classified as abnormal if they occurred spontaneously, lasted longer than 30 sec, or were repetitive. In 7 of the patients abnormal sphincteric contraction was found; in 2, sphincteric contraction was disturbed by spontaneous contractions, and in 3 by repetitive and/or prolonged post-deglutitive contractions. The frequency of prolonged contractions increased progressively from proximal to distal esophagus, while repetitive contractions were not correlated with site. Nine patients showed abnormal spontaneous motor activity. One had vigorous achalasia, 5 had diffuse esophageal spasm, and 6 did not adhere to any previously defined pattern. It is concluded that an underlying esophageal motor dysfunction be suspected in patients with mid-thoracic esophageal diverticula.

1928 OESOPHAGEAL ACID CLEARING: ONE FACTOR IN THE PRODUCTION OF REFLUX OESOPHAGITIS. *(E.)* Stanciu, C.; Bennett, J. R. (Hull R. Infirm., Kingston upon Hull, England). *Gut* 15(11):852-857, 1974.

Esophageal acid clearing, 1 factor in the production of reflux esophagitis, was studied in 89 subjects, who fell into 5 groups diagnostically. There were: 14 subjects without reflux symptoms; 4 patients with hiatal hernias but no reflux symptoms; 11 patients with duodenal ulceration and reflux symptoms; 55 patients with reflux symptoms (12 had hiatal hernia and 18 had gastroesophageal reflux shown radiologically); and 5 patients with esophageal motor abnormalities (2 achalasia and 3 diffuse esophageal spasm). Esophageal acid clearing was measured by counting the number of swallows needed to raise the pH of the lower esophagus from 1.5-5.0 after instillation of 15 ml 0.1 N hydrochloric acid. Normal subjects and those asymptomatic patients with hiatal hernias had a result less than 12. Patients with reflux symptoms and those with esophageal motility disorders had abnormal acid clearance (mean number of swallows greater than 12). Of the 20 patients with abnormal esophageal motility, 18 had prolonged acid clearing, however many patients with delayed clearing had no abnormal motility. As the degree of esophagitis increased, acid clearing deteriorated. Acid clearance correlated well with the mean duration of spontaneous episodes of acid reflux during 15-hr continuous recordings of lower esophageal pH. Abnormal acid clearance was improved by raising the bedhead, by medical treatment for esophagitis, and by i.v. metoclopramide. Abnormal acid clearing may result from disturbed esophageal motility and render these patients susceptible to reflux esophagitis. However, gastroesophageal reflux may lead to impaired acid clearance, by creating abnormal motility and by effects on the esophageal wall, and thus lead to a 'vicious-spiral' esophagitis. Acid clearance must be improved when treating gastroesophageal reflux.

1929 MOTILITY STUDIES IN FIFTY PATIENTS WITH ACHALASIA OF THE ESOPHAGUS. *(E.)* Uribe, Jr., P.; Csendes, A.; Larraín, A.; Ayala, M. (Jose Joaquin Aquirre Hosp.), Univ. Chile, Santiago). *Am. J. Gastroenterol.* 62(4):333-336, 1974.

Esophageal motility studies were performed on 50 patients with achalasia and 30 normal controls. Patients were intubated through the mouth while in a supine position, with continuously perfused polyethylene catheters. The catheters were withdrawn from fundus to thorax, taking periodic 10 sec pressure readings. The achalasia patients had a resting gastroesophageal pressure of 19.0 mm Hg, significantly higher than control values (11.0). On swallowing, the percentage of sphincter relaxation was 52.6% as compared to 100% for the controls. The intraesophageal resting pressure was 6.1 in the achalasia patients and -3.5 in the controls. This confirms previous findings that relaxation of the gastroesophageal sphincter does occur during swallowing in achalasia, and that hypertensive gastroesophageal sphincter also occurs in this condition.

1930 IMPROVEMENT IN LOWER ESOPHAGEAL SPHINCTER PRESSURE FOLLOWING SURGERY FOR COMPLICATED GASTROESOPHAGEAL REFLUX. *(E.)* Dimarino, A. J.; Rosato, E.; Rosato, F.; Cohen, S. (Hosp. Univ. Pennsylvania, Philadelphia). *Ann. Surg.* 181(2):239-242, 1975.

A comparison was made of the pre- and postoperative lower esophageal sphincter (LES) pressures in nine patients undergoing a posterior gastropexy for complicated gastroesophageal reflux. Ten age-matched controls were also evaluated. Water-filled polyvinyl catheters (1.4 mm internal diameter) were used to transmit intra-luminal pressures to external transducers. Pressures from esophagus, LES, and stomach were simultaneously recorded. LES pressure was increased from 4.4 to 13.9 mm Hg following surgery. The ratio of the change in LES pressure compared to the change in gastric pressure during increases in intra-abdominal pressure ($\Delta S:\Delta G$) was 0.59 preoperatively and 0.94 postoperatively. All patients were asymptomatic after surgery. Although the resting LES pressure and the $\Delta S:\Delta G$ ratio following surgery were increased, they remained significantly less than comparable values in the controls. The results suggest that the clinical improvement following surgery for gastroesophageal reflux may be due to the increase in resting LES pressure and the improved response of the lower esophageal sphincter to increased intra-abdominal pressure.

1931 EPIDEMIOLOGY OF CANCER OF THE OESOPHAGUS IN INDIA. INCIDENCE, MORTALITY, DISTRIBUTION. *(E.)* Gangadharan, P. (Tata Mem. Hosp., Bombay, India). *Indian J. Surg.* 36(8):293-298, 1974.

1932 CANCER SUPERIMPOSED ON MEGAESOPHAGUS. *(It.)* Coloni, G. F.; Martelli, M.; Mini, A.; Ricci, C. (Inst. 2nd Surg. Clin., Univ. Rome, Italy). *Policlinico [Chir.]* 80(3):119-141, 1973.

1933 GRANULAR CELL MYOBLASTOMA: ANOTHER UNUSUAL ESOPHAGEAL LESION. *(E.)* Calhoun, T.; Odelowo, E. O. O.; Ali, S.; Kurtz, L. H.; Simmons, R. H. (Howard Univ. Coll. Med., Washington, D.C.). *J. Thorac. Cardiovasc. Surg.* 69(3):472-475, 1974.

1934 MANAGEMENT OF OESOPHAGEAL CARCINOMA. A REPORT OF 267 CASES. *(E.)* Inberg, M. V.; Scheinin, T. M.; Voutilainen, A.; Havia, T.; Nikkanen, T. A. V. (Dep. Surg., Univ. Turku, Finland). *Scand. J. Thorac. Cardiovasc. Surg.* 3(8):220-227, 1974.

1935 A CRITICAL EVALUATION AND RATIONALE OF PREOPERATIVE RADIATION IN THE TREATMENT OF CANCER OF THE MIDDLE THORACIC OESOPHAGUS: AN EXPERIENCE BASED ON 100 RESECTIONS. *(E.)* Desai, P. B. (Tata Mem. Hosp., Bombay, India). *Indian J. Surg.* 36(8):309-311, 1974.

1936 MODERN TREATMENT FOR CARCINOMAS OF THE ESOPHAGUS, STOMACH, AND DUODENUM. *(Ger.)* Bunte, H. (Surg. Clin., Univ. Munster, Germany). *Therapiewoche* 24(17):1902-1910, 1974.

1937 SURGERY FOR CANCER OF THE OESOPHAGUS. *(E.)* Mehta, M. J.; Limaye, S. M. (B.J. Med. Coll., Poona, India). *Indian J. Surg.* 36(8): 312-318, 1974.

1938 LESSONS LEARNT IN OESOPHAGEAL SURGERY FOR CANCER—A PERSONAL EXPERIENCE OF OVER 15 YEARS AND 300 RESECTIONS. *(E.)* Desai, P. B. (Tata Mem. Hosp., Bombay, India). *Indian J. Surg.* 36(8): 299-308, 1974.

1939 OESOPHAGEAL CANCER. *(E.)* Gupta, R. S. (Holy Fam. Hosp., New Delhi, India). *Indian J. Surg.* 36(8):319-322, 1974.

1940 ETIOLOGY AND PATHOGENESIS OF MALLORY-WEISS SYNDROME. *(Rus.)* Bellmann; Wohlgemuth, B.; Kothe, W.; Brand, G.; Haupt, R.; Scheuner, G.; Hutschenreiter, J.; Rotzscher, K. (no affil.). *Khirurgiia (Mosk.)* (2):14-19, 1974.

1941 THERAPEUTIC PROBLEMS IN MASSIVE BLEEDING FROM ESOPHAGEAL VARICES. *(Ger.)* Brunner, H.; Kurock, W.; Ehlert, C. P. (Surg. Clin., Univ. Mainz, Germany). *Therapiewoche* 24(18):1994-2002, 1974.

1942 ENDOSCOPIC FIBROSCLEROSIS OF ESOPHAGEAL VARICES: INDICATIONS, COMPLICATIONS, AND SEQUELAE. *(Ger.)* Paquet, K. J. (Surg. Clin., Rhenish Friedrich Wilhelms Univ., Bonn, Germany). *Therapiewoche* 24(5):381-382, 385, 1974.

1943 SCLEROSING INJECTION OF ESOPHAGEAL VARICES THROUGH TRANSHEPATIC SELECTIVE CATHETERIZATION OF THE GASTRIC CORONARY VEIN. A PRELIMINARY REPORT. *(E.)* Lunderquist, A.; Vang, J. (Univ. Hosp., Lund, Sweden). *Acta Radiol.* 15(5):546-550, 1974.

1944 ESOPHAGEAL ATRESIA WITH DISTAL TRACHEO-ESOPHAGEAL FISTULA (EXPERIENCE WITH 8 CONSECUTIVE CASES). *(E.)* Slim, M. S. (Sch. Med., American Univ. Beirut, Lebanon). *Leban. Med. J.* 27(4):451-462, 1974.

1945 THE EFFECT OF POLYPEPTIDE HORMONES ON THE LOWER ESOPHAGEAL SPHINCTER IN ACHALASIA. *(Ger.)* Siewert, R.; Fruh, E.; Waldeck, F.; Schmidt, H. (Clin. Gen. Surg., Univ. Gottingen, Germany). *Z. Gastroenterol.* 12(2):117-120, 1974.

1946 LONG-TERM CLINICAL, RADIOLOGICAL AND MANOMETRIC FOLLOW-UP OF PATIENTS WITH ACHALASIA TREATED WITH PNEUMATIC DILATATION. *(E.)* Csendes, A.; Strauszer, T. (Jose Joaquin Aguirre Hosp., Univ. Chile, Santiago). *Digestion* 11(1/2):128-134, 1974.

1947 AN ATTEMPT TO INFLUENCE THE ESOPHAGEAL MOTILITY BY HYPNOSUGGESTION. *(E.)* Cerny, M.; Lukasova, A.; Pitha, F.; Setka, O. (Med. Sch., Charles Univ., Prague, Czechoslovakia). *Act. Nerv. Super. (Praha)* 16(2):123, 1974.

1948 SCLEROSIS OF THE ESOPHAGEAL WALL: PRELIMINARY RESULTS. *(Ger.)* Buess, H. J.; Kapp, F. (Med. Clin., Univ. Basel, Switzerland). *Z. Gastroenterol.* 12(1):25-26, 1974.

1949 ESOPHAGEAL STRICTURE COMPLICATING STEVENS-JOHNSON SYNDROME. A CASE REPORT. *(E.)* Stein, M. R.; Thompson, Jr., C. K.; Sawicki, J. E.; Martel, A. J. (Letterman Army Med. Cent., San Francisco, Calif.). *Am. J. Gastroenterol.* 62(5):435-439, 1974.

1950 NONSPECIFIC MEDIASTINAL LYMPHADENITIS ASSOCIATED WITH COMPRESSION OF THE MIDDLE THIRD OF THE ESOPHAGUS. *(E.)* McLoud, T. C.; Putman, C. E. (Yale Univ. Med. Sch., New Haven, Conn.). *Am. J. Dig. Dis.* 19(12):1162-1164, 1974.

1951 HIATAL HERNIA AND GASTROESOPHAGEAL REFLUX IN INFANTS AND CHILDREN: ANALYSIS OF THE INCIDENCE IN NORTH AMERICAN CHILDREN. *(E.)* Darling, D. B.; Fisher, J. H.; Gellis, S. S. (New Engl. Med. Cent. Hosp., Boston, Mass.). *Pediatrics* 54(4):450-455, 1974.

1952 TWENTY-FOUR-HOUR pH MONITORING OF THE DISTAL ESOPHAGUS. A QUANTITATIVE MEASURE OF GASTROESOPHAGEAL REFLUX. *(E.)* Johnson, L. F.; Demeester, T. R. (Univ. Hawaii Sch. Med., Honolulu). *Am. J. Gastroenterol.* 62(4):325-332, 1974.

1953 CAN SYMPTOMS OR RADIOGRAPHIC REFLUX BE PREDICTED BY MEASURING CARDIAC SPHINCTER PRESSURE? *(E.)* Dilawari, J. B.; Edwards, D. A. W.; Girmes, D. H. (Cent. Middlesex Hosp., London, England). *Gut* 15(10):832-833, 1974.

ESOPHAGUS

1954 MANOMETRIC ASSESSMENT OF OESOPHAGEAL FUNCTION AFTER THE NISSEN FUNDOPLICATION. *(E.)* Bremner, C. G. (Dep. Surg., Univ. Witwatersrand, Johannesburg, South Africa). *S. Afr. J. Surg.* 12(2): 73, 1974.

1955 HERPES VIRUS ESOPHAGITIS, CONSISTENT WITH HERPES SIMPLEX, VISUALIZED ENDOSCOPICALLY. *(E.)* Klotz, D. A.; Silverman, L. (Hoag Presbyt. Hosp., Newport Beach, Calif.). *Gastrointest. Endosc.* 21(2):71-73, 1974.

1956 CHANGES IN THE ESOPHAGUS IN 1,000 AUTOPSY SPECIMENS. *(E.)* Postlethwait, R. W.; Musser, A. W. (VA Hosp., Durham, N.C.). *J. Thorac. Cardiovasc. Surg.* 68(6):953-956, 1974.

1957 ESOPHAGUS, STOMACH, AND DUODENUM: MEDICAL THERAPY. *(Ger.)* Gerlach, U.; van Husen, N. (Med. Clin., Univ. Munster, Germany). *Therapiewoche* 24(17):1889-1895, 1974.

See also, 1655, 1663, 1666, 1891, 1892, 1893, 1894, 2270, 2315, 2331.

STOMACH

1958 MULTIPLE GASTROINTESTINAL SCHWANNOMAS (VON RECKLINGHAUSEN'S DISEASE). A FAMILIAL CASE. *(Fr.)* Rosner, D.; Hivet, M.; Lagadec, B.; Conte, J. (St. Antoine Hosp., Paris, France). *Med. Chir. Dig.* 2(1):39-44, 1973.

A 37-yr-old man had been hospitalized earlier for melena, had undergone gastro-duodenal transit, had been put on anti-ulcer treatment for a mid-duodenal ulcer, and had undergone 3 more episodes of melena and a normal i.v. cholangiograph had been taken. An 80 cm length of small intestine afflicted with schwannomas was resected, leaving some small tumors up to the terminal ileum. Multiple endo- and exointestinal nodular tumors were attached to the resected intestines. The prognosis was somewhat uncertain because of the multiplicity of the neurinomatous foci and because the largest, ulcerated tumor possibly represented the homolog of the "royal tumors" of von Recklinghausen's disease with subcutaneous symptoms, but 1 yr later the patient was still well. The pre-operative symptoms showed few signs of von Recklinghausen's disease: groups of spots resembling cafe au lait on the palms of the hands, the soles of the feet, and in the perianal region; lacunary images on x-rays of the mid-duodenal ulcer and the large ulcerated tumor. The patient's mother had died 10 yr earlier following surgery for hematemesis related to a degenerated gastric schwannoma. She also showed cutaneous signs of von Recklinghausen's disease. One of the patient's 2 sisters had been operated on twice for hematemesis, and the second time revealed an ulcerated tumor and numerous schwannomas along the small intestine. She also had the skin spots. The patient's 12-yr-old daughter showed cutaneous symptoms and digestive problems, but his 11-yr-old son had no symptoms. His first sister's 17-yr-old son had cutaneous signs and digestive problems, but her 2 younger children showed no symptoms. The patient's second sister's 11-yr-old daughter showed cutaneous signs and the intestines were suspect. Her 2 brothers showed no symptoms. This family study further adds to the literature on von Recklinghausen's disease.

1959 PATHOPHYSIOLOGY OF STRESS ULCER AND ITS PREVENTION. II. PROSTAGLANDIN E_1 AND MICROCIRCULATORY RESPONSES IN STRESS ULCER. *(E.)* Kawarada, Y.; Lambek, J.; Matsumoto, T. (Hahnemann Med. Coll. Hosp., Philadelphia, Pa.). *Am. J. Surg.* 129(2):217-222, 1975.

The effects of prostaglandin E_1 in prevention of stress ulcers were investigated in the rat. Male Wistar rats (330) were fasted for 24 hr and most were subjected to stress by restraint for 2 hr in a conduit in a room at 4 C. Rats were divided into 10 groups: 5 groups had no prostaglandin or restraint; 5 had their stomachs inflated with air, but no prostaglandin or restraint; 5 were given 5 mg/kg prostaglandin s.c. but no restraint; 1 was restrained but given no prostaglandin; and the remaining 6 groups were restrained and given dosages of 0.5 mg/kg, 1.0 mg/kg, 2 mg/kg, 3 mg/kg, 4 mg/kg and 5 mg/kg prostaglandin s.c., resp. In 5 rats of each group, a silicone rubber compound was perfused through the gastric and duodenal arteries, capillaries, and veins. Acute gastric ulcers developed in 90% of restrained animals given no prostaglandin; 87% of those given 0.5 mg/kg; 65% given 1.0; 43% given 2.0; 17% given 3.0; and 0% given 4.0 and 5.0. The severity of the ulcers decreased along with the incidence. With increasing dosages, however, there were increasing symptoms of diarrhea, loss of activity, labored breathing, and increased nasal secretion. The mortality rates among the preceding 7 groups were 0%, 3%, 5%, 7.5%, 15%, 30%, 43%, and 56% resp. Comparison of the control group with the group with stomach distention showed the latter had thinner mucosa, muscle layers, and serosa, and narrowing of blood vessels. In the group given 5 mg/kg prostaglandin and no restraint, the mucosa, muscle layers, and serosa were stretched, but vasodilation could not be evaluated. In the group given restraint and no prostaglandin, the silicone rubber did not fill the capillaries well; avascular areas, necrosis, and blood clots were found. In the groups given 0.5 and 1.0 mg/kg, findings were similar to the previous group. In those given 2-3 mg/kg, the stomach wall layers were moderately stretched, and arterioles and collecting veins were narrow. In those given 4-5 mg/kg, the walls were extremely distended, and the vessels were moderately

narrowed. It is concluded that prostaglandin at the 4-5 mg/kg dose prevents stress ulceration, but complications and mortality rates were higher than 40% at these doses.

1960 RELATION BETWEEN MALIGNANT TRANSFORMATION OF POLYP-LIKE ADENOMAS OF THE STOMACH AND THEIR FORM AND SIZE. *(Rus.)* Lukinykh, A. K. (Ryazan Med. Inst., USSR). *Klin. Med. (Mosk.)* 52(11): 58-62, 1974.

1961 SUBCLASSIFICATION OF EARLY STOMACH CANCER. *(Ger.)* Hermanek, P.; Rosch, W. (Surg. Clin., Univ. Erlangen, Germany). *Verh. Dtsch. Ges. Pathol.* 57:447, 1973.

1962 GASTRIC LOCALIZATION OF MULTIPLE MYELOMA. *(Fr.)* Bourreille, J.; Hemet, J.; Tayot, J.; Maitre, A. M. (Univ. Hosp. Cent., Rouen, France). *Sem. Hop. Paris* 50(25):1737-1742, 1974.

1963 GASTRIC TERATOMA IN INFANCY AND CHILDHOOD: REPORT OF THREE CASES AND REVIEW OF LITERATURE. *(E.)* Ohgami, H.; Ikeda, K.; Koga, Y.; Suita, S.; Nagasaki, A.; Hayashida, Y. (Kyushu Univ. Fac. Med., Fukuoka, Japan). *Jpn. J. Surg.* 3(4):218-228, 1973.

1964 GASTRIC HEMANGIOMA. *(E.)* Toffler, R. B.; Osborn, D. (Yale Univ. Sch. Med., New Haven, Conn.). *Am. J. Gastroenterol.* 62(5):448-452, 1974.

1965 IMMUNOFLUORESCENT STAINING OF PLACENTAL ALKALINE PHOSPHATASE IN GASTRIC AND RECTAL CARCINOMA. *(E.)* Kawasaki, H.; Takeuchi, M.; Kimoto, E. (Kurume Univ. Sch. Med., Japan). *GANN* 65(6):473-479, 1974.

1966 INTEGRATION OF CHEMOTHERAPY INTO COMBINED MODALITY TREATMENT OF SOLID TUMORS. III. GASTRIC CANCER. *(E.)* Comis, R. L.; Carter, S. K. (Natl. Cancer Inst., Bethesda, Md.). *Cancer Treat. Rev.* 1(3):221-238, 1974.

1967 INTRAOPERATIVE RADIOTHERAPY OF GASTRIC CANCER. *(E.)* Abe, M.; Yabumoto, E.; Takahashi, M.; Tobe, T.; Mori, K. (Fac. Med., Kyoto Univ., Japan). *Cancer* 34(6):2034-2041, 1974.

1968 LACTATE DEHYDROGENASE ISOENZYMES IN THE GASTRIC MUCOSA OF PATIENTS WITH CHRONIC GASTRITIS ACCOMPANIED BY HYPOCHLORHYDRIA AND STOMACH CANCER. *(Rus.)* Lomachenkov, V. D.; Aksenova, O. F. (Smolensk Med. Inst., USSR). *Klin. Med. (Mosk.)* 52(11):47-52, 1974.

1969 ATROPHIC GASTRITIS AND ITS SEQUELAE: EXPERIENCE FROM A 19-23 YEAR FOLLOWUP. *(Cz.)* Siurala, M.; Lehtola, J.; Ihamaki, T. (no affil.). *Cesk. Gastroenterol. Vys.* 28(2):106-109, 1974.

1970 SERUM-GASTRIN, VITILIGO, AND ACHLORHYDRIC ATROPHIC GASTRITIS. *(E.)* Howitz, J.; Rehfeld, J. F. (Dep. Dermatol., Finsen Inst., Copenhagen, Denmark). *Lancet* 2(7893):1399-1400, 1974.

1971 STUDY OF GASTRIC EMPTYING OF WATER AND A 10% GLUCOSE SOLUTION IN CHRONIC ATROPHIC GASTRITIS. *(Sp.)* Rodrigo Gomez, J. M.; Aparisi Quereda, L.; Serra Desfilis, M. A.; Guix Garcia, J.; Camps Vilata, B.; Olmo Puchalt, J.; Wassel, A.; Garcia-Conde Gomez, F. J. (Fac. Med., Valencia, Spain). *Rev. Esp. Enferm. Apar. Dig.* 44(5):457-468, 1974.

1972 EROSIVE GASTRITIS WITH PROTEIN EXUDATION. *(Ger.)* Filippini, L.; Isliker, K. (Canton. Hosp., Lucerne, Switzerland). *Z. Gastroenterol.* 12(1):44, 1974.

1973 REFLUX BILE GASTRITIS. *(E.)* Thorfinnson, P. C.; Brow, J. R. (Sunnybrook Med. Cent., Univ. Toronto, Canada). *J. Can. Assoc. Radiol.* 25 (4):263-268, 1974.

1974 THE IMPORTANCE OF AN INNERVATED INTACT ANTRUM IN PREVENTING SYMPTOMATIC DUODENO-GASTRIC REFLUX. *(E.)* Keighley, M. R. B.; Asquith, P.; Hoare, A.; Alexander-Williams, J. (Gen. Hosp., Birmingham, England). *Gut* 15(10):836, 1974.

1975 GASTROCOLIC FISTULA, A RARE COMPLICATION OF BENIGN GASTRIC ULCER: REPORT OF A CASE. *(E.)* Smith, D. L.; Comer, T. P. (Kern Gen. Hosp., Bakersfield, Calif.). *Dis. Colon Rectum* 17(6):769-770, 1974.

1976 PYLORIC CHANNEL ULCER WITH BETAZOLE-FAST ACHLORHYDRIA. *(E.)* Korn, E. R.; Foroozan, P. (Scripps Clin. Res. Found., La Jolla, Calif.). *Gastroenterology* 67(6):1248-1249, 1974.

1977 EFFECT OF COMPOUND 561 ON GASTRIC ULCERS. *(E.)* Slovak, A.; Svatos, A. (no affil.). *Manuf. Chem. Aerosol News* 45(12):44-45, 1974.

1978 GASTRODUODENAL ULCERS IN DOGS PRODUCED BY ELECTRIC STRESS AFTER RESERPINIZATION. *(E.)* Hara, M. (Nihon Univ. Sch. Med., Japan). *Nihon Univ. J. Med.* 16(2):119-128, 1974.

1979 SOLITARY GASTRIC VARIX. *(E.)* Williams, D. R.; Paull, A.; Grant, A. K.; Tucker, W. G. (Queen Elizabeth Hosp., Sydney, Australia). *Australas. Radiol.* 18(2):183-186, 1974.

1980 VASCULAR DISEASE OF THE SUBMUCOSA IN THE STOMACH AND SMALL INTESTINE AS A CAUSE OF PROFUSE BLEEDING: LITERATURE REVIEW. *(Rus.)* Belousova, I. M. (Clin. Hosp. No. 6, Moscow, USSR). *Khirurgiia (Mosk.)* (11):132-140, 1974.

1981 LONG-TERM EFFECTS OF TRUNCAL, SELECTIVE, AND HIGHLY SELECTIVE VAGOTOMY ON GASTRIC SECRETION IN MAN. *(E.)* Greenall, M. J.; Lyndon, P. J.; Goligher, J. C.; Johnston, D. (Gen. Infirm., Leeds, England). *Gut* 15(10):834, 1974.

1982 GASTRIC EMPTYING IN THE IVOR LEWIS OESOPHAGOGASTROSTOMY. *(E.)* Hinder, R. A. (Dep. Surg., Univ. Witwatersrand, Johannesburg, South Africa). *S. Afr. J. Surg.* 12(2):73, 1974.

1983 GASTRIC EMPTYING RATE MEASUREMENT IN MAN: A METHOD FOR SIMULTANEOUS STUDY OF SOLID AND LIQUID PHASES. *(E.)* Heading, R. C.; Tothill, P.; McLoughlin, G. P.; Shearman, D. J. C. (R. Infirm. Edinburgh, Scotland). *Gut* 15(10):841, 1974.

1984 LONG-TERM RESULTS FROM EXTENSIVE PANCREATECTOMY AND GASTRECTOMY. *(Rus.)* Gusarev, V. F.; Ganza, V. T. (M. Gorky Inst. Postgrad. Med., Zaporozhe, USSR). *Klin. Khir.* (9):80-81, 1974.

1985 THE DIAGNOSIS OF ANASTOMOTIC INFLAMMATION AND PANCREATITIS FOLLOWING GASTRECTOMY. *(Rus.)* Glivenko, Iu. A.; Myrkin, S. D. (Crimean Med. Inst., Simferopol, USSR). *Klin. Khir.* (9): 79-80, 1974.

1986 CHANGES IN PORTAL BLOOD COMPOSITION IN OPERATIONS ON THE GASTROINTESTINAL TRACT. *(Rus.)* Zavgorodnii, L. G.; Gnilitskaia, L. I.; Taptapiants, A. A. (Donetsk Med. Inst., USSR). *Klin. Khir.* (9):63-64, 1974.

1987 ROLE OF GASTRIN IN HYPERSECRETORY DISORDERS IN MAN. *(E.)* McGuigan, J. E. (Coll. Med., Univ. Florida, Gainesville). *Annu. Rev. Med.* 25: 233-249, 1974.

1988 PROVOKING GASTRIN RELEASE — WHICH STIMULUS? *(E.)* Fielding, L. P.; Russell, R. C. G.; Bloom, S. R. (St. Mary's Hosp., London, England). *Gut* 15(10):835, 1974.

1989 PROSTAGLANDINS AND THEIR EFFECT ON GASTROINTESTINAL FUNCTION. *(Rus.)* Ilinich, V. K. (N. N. Burdenko Clin. Mil. Hosp., Moscow, USSR). *Klin. Med. (Mosk.)* 52(1):30-34, 1974.

1990 ENZYME HISTOCHEMICAL INVESTIGATIONS OF HETEROTOPIC GASTRIC EPITHELIUM IN THE DUODENUM. *(E.)* Johansen, A. (Bispebjerg Hosp., Copenhagen, Denmark). *Acta Pathol. Microbiol. Scand. [A]* 82(5):613-617, 1974.

1991 STUDY OF LACTATE DEHYDROGENASE ISOENZYMES IN NORMAL AND HYPERTROPHIC PYLORIC MUSCLE. *(Sp.)* Martinez, E. (no affil.). *Rev. Esp. Enferm. Apar. Dig.* 44(5):485-492, 1974.

1992 IS GASTROESOPHAGEAL PROLAPSE RESPONSIBLE FOR CHANGES IN THE GASTRIC MUCOSA? *(Ger.)* Miller, G.; Savary, M. (Munic. Hosp., Solothurn, Switzerland). *Z. Gastroenterol.* 12(1):38, 1974.

1993 GASTRIC INFARCTION. *(E.)* Kerstein, M. D.; Goldberg, B.; Panter, B.; Tilson, M. D.; Spiro, H. (Yale Univ. Sch. Med., New Haven, Conn.). *Gastroenterology* 67(6):1238-1239, 1974.

1994 GIANT HYPERTROPHY OF THE GASTRIC RUGAE AND MENETRIER'S DISEASE. A SEARCH IN SYDNEY. *(E.)* Broadfoot, E. (Sydney Hosp., Australia). *Australas. Radiol.* 18(2):179-182, 1974.

1995 GASTRIC OUTLET OBSTRUCTION — AN UNUSUAL CAUSE. *(E.)* Furtado, M. A. (Bundaberg Gen. Hosp., Australia). *Australas. Radiol.* 18(2): 187-190, 1974.

1996 SILENT TRICHOBEZOAR. A CASE REPORT. *(E.)* Winston, D.; Katz, S.; Katzka, I. (North Shore Univ. Hosp., Manhasset, N.Y.). *Am. J. Gastroenterol.* 62(4):367-370, 1974.

1997 VAGOTOMY AND BEZOARS. *(Ger.)* Miller, G.; Clemencon, G. (Munic. Hosp., Solothurn, Switzerland). *Z. Gastroenterol.* 12(1):33, 1974.

1998 ELECTRON MICROSCOPE STUDY ON THE PATHOGENESIS OF UREMIC STOMACH DISEASE. *(Ger.)* Kreinsen, U.; Krempien, B.; Bauer, V. H.; Ritz, E. (Pathol. Inst., Univ. Heidelberg, Germany). *Verh. Dtsch. Ges. Pathol.* 57:451, 1973.

1999 ENDOSCOPY OF THE GASTRIC CARDIA. *(Ger.)* Heinkel, K.; Schurholz, J. (Bad Cannstatt Hosp., Stuttgart, Germany). *Z. Gastroenterol.* 12 (1):37, 1974.

See also, 1727, 1891, 1892, 1894, 1895, 1896, 1897, 1898, 1936, 1957, 2010, 2016, 2023, 2028, 2029, 2058, 2298, 2331, 2334.

2000 SOME EARLY CHARACTERISTICS OF PEPTIC ULCER DECEDENTS. *(E.)* Polednak, A. P. (State Dep. Health, Albany, N.Y.). *Gastroenterology* 67(6): 1094-1100, 1974.

A number of early characteristics were analyzed among 163 peptic ulcer decedents (79 gastric and 84 duodenal ulcer) and 163 age-matched control subjects. Data were obtained while the men were at Harvard University, at a mean age of 20 yr. Data were analyzed by matched pairs, the degree of association between the antecedent factor and the disease being evaluated by an odds ratio. There was no higher frequency of pulmonary disease (pneumonia, bronchitis, pleurisy, or asthma) in ulcer decedents as compared to controls. Both gastric and duodenal ulcer patients had lost more fathers than did controls, however duodenal ulcer cases had lost fewer mothers than controls. Palpitations, insomnia, and skin eruptions were reported more frequently by ulcer patients, while dyspepsia was not reported more frequently. These differences could reflect early life experiences or basic personality of ulcer-prone individuals, but additional studies must be done to clarify the role of such experiences or personality characteristics in ulcer pathogenesis.

2001 HYPOVITAMINOSIS B_{12} FOLLOWING PARTIAL GASTRECTOMY BY THE BILLROTH II METHOD. *(E.)* Rygvold, O. (Ostfold Cent. Hosp., Fredrikstad, Norway). *Scand. J. Gastroenterol.* [*Suppl.*] 9(29):57-64, 1974.

Hypovitaminosis B_{12} following 2/3 partial gastrectomy by the Billroth II method was studied in 351 patients who were followed for 1-23 yr post-operatively. In 143 the surgery had been done for gastric ulcer, and in 208 for duodenal ulcer. Post-operative antianemic therapy had not been given. Vitamin B_{12} deficiency was found in 20%, with the incidence in the gastric ulcer group being twice as high as in the duodenal ulcer group. In 16% the serum B_{12} level was less than 150 pg/ml, and 16% had levels between 150 and 200 pg/ml. The regression of serum B_{12} was most marked during the first 8-10 years after surgery, then it leveled off. About 1/2 the patients with B_{12} deficiency had normal hemoglobin levels, and 1/3 of the deficient cases had iron deficiency also. Hypovitaminosis B_{12} combined with anemia was most often found in gastric ulcer patients and females. Patients with gastritic changes at the time of surgery and those with the smallest gastric stumps (according to radiographic assessment) had a greater preponderance of B_{12} deficiency. According to the Schilling tests, the hypovitaminosis B_{12} was due to intestinal malabsorption syndromes in 67% of those with serum B_{12} levels below 150 pg/ml, and in 69% of those in the 150-200 pg/ml range. In 32% the hypovitaminosis was due to deficiency of the intrinsic factor. The dumping syndrome and milk dyspepsia were the only types of post-operative dyspepsia correlated to some extent to the vitamin deficiency. Because of the high incidence of hypovitaminosis B_{12} patients with this type of operation, the author recommended prophylactic B_{12} and iron supplements.

2002 THE EFFECT OF POSTURE ON THE GASTRIC EMPTYING OF SOLID MEALS IN NORMAL SUBJECTS AND PATIENTS AFTER VAGOTOMY. *(E.)* Hancock, B. D.; Bowen-Jones, E.; Dixon, R.; Testa, T.; Dymock, W.; Cowley, D. J. (Univ. Hosp. South Manchester, England). *Br. J. Surg.* 61(12):945-949, 1974.

The effect of posture on the gastric emptying rate of solid meals was studied in normal subjects and in patients after a truncal vagotomy and pyloroplasty or after a highly selective vagotomy. Ten healthy subjects and 10 vagotomy patients (within 3 months of surgery) were studied. A solid meal was labeled with ^{51}Cr and the rate of emptying measured with a gamma camera. In normal subjects, posture did not affect the rate of emptying, but in patients who had had a truncal vagotomy and pyloroplasty (total of 5 patients), the stomach emptied more rapidly when standing than when lying down. When lying down the rate of emptying was significantly less than in normal subjects, but normal when standing. In the 5 patients who had had a highly selective vagotomy the stomach emptied more rapidly when standing than when lying down, but when lying down the rate was normal. When standing the rate was significantly faster than that in normal subjects. Thus posture had a very striking effect on the rate of gastric emptying of solids after both types of vagotomy, but had no effect on emptying in normal subjects. There was no difference in the initial emptying rate between the 2 groups studied, which is in contrast to some previous studies.

2003 EFFECT OF RISING INTRAGASTRIC pH INDUCED BY SEVERAL ANTACIDS ON SERUM GASTRIN CONCENTRATIONS IN DUODENAL ULCER PATIENTS AND IN A CONTROL GROUP. *(E.)* Feurle, G.E. (Med. Polyclin., Univ. Heidelberg, Germany). *Gastroenterology* 68(1):1-7, 1975.

The effects of 3 antacids (magnesium hydroxide, aluminium hydroxide, and calcium carbonate) on serum gastrin levels and intragastric pH were compared with the effects of the corresponding nonbuffering chloride compounds (magnesium chloride, aluminium chloride, and calcium chloride) in duodenal ulcer patients and in a control group. Patients with clinical and radiological evidence of duodenal ulcer (52) and 9 controls without gastrointestinal disease were used. No significant rise of serum gastrin levels occurred after $BaSO_4$ was given to assess the contribution of intragastric volume. Intragastric pH and serum gastrin, determined radioimmunologically, increased in patients after $Mg(OH)_2$ was given. Serum gastrin also increased when the chloride analogues were given to each group, except after $MgCl_2$ in the control group. All the antacids caused a rise in serum gastrin in all groups and this rise was significantly higher than after the chlorides in most groups with most of the compounds. Serum gastrin concentrations rose significantly whenever intragastric pH was raised, and also after $MgCl_2$, $AlCl_3$, and $CaCl_2$ with a pH of 2, when the pH was not significantly altered. In control subjects, gastrin response was smaller than in the duodenal ulcer patients. Serum gastrin levels after $CaCl_2$ (pH 10) rose as much as after calcium

carbonate, whereas after $CaCl_2$ (pH 2) the rise was much lower. The results suggest that administration of nonbuffering Mg-, Al-, and Ca-chlorides lead to elevated serum gastrin levels in duodenal ulcer patients, and rising intragastric pH exerts an additional gastrin response.

2004 INTRAVENOUS PENTAGASTRIN AS A PARTIAL AGONIST OF GASTRIC SECRETION IN MAN: EVIDENCE IN FAVOR OF THE EXISTENCE OF HORMONAL INHIBITORY SITES. *(E.)* Prugh, M. F.; Schorr, B. A.; Vlahcevic, Z. R.; Makhlouf, G. M. (Med. Coll. Virginia, Richmond). *Gastroenterology*, 68(1):45-49, 1975.

The gastric secretory response to prompt i.v. injection of pentagastrin (0.125-0.75 µg/kg) was investigated in 6 male subjects (3 with duodenal ulcer and 3 normal). Each subject was tested with histamine acid phosphate (40 µg/kg s.c.). After injection with pentagastrin, the acid and pepsin concentrations rose to a peak in about 5 min and then declined to control levels in 30-40 min. The concentrations were higher in duodenal ulcer patients. The highest responses to pentagastrin and histamine were correlated. Sensitivity to pentagastrin, expressed in the D_{50}, was higher in duodenal ulcer patients. Unexpectedly, the highest response to pentagastrin was only 40% of the peak response to the s.c. histamine. Thus, i.v. pentagastrin acted as a partial agonist (i.e., that with this mode of administration a maximal response could not be attained) with an efficacy of about 0.4. The effect of prompt pentagastrin injection contrasted with the effect of slow i.v. infusion of the substance, as well as with the effects of gastrin-17 given either by prompt injection or slow infusion. The authors proposed that the secretory receptor for gastrin and its analogues consists of 2 sites: a high affinity stimulatory site and a low affinity inhibitory site. Partial agonism after prompt i.v. injection would result from blood and tissue transients which are successively too high (partially inhibitory) and low (stimulatory). The relative affinities for the 2 sites could explain the disparate effects of analogues and species differences.

2005 INTERACTION OF CHOLINERGIC FUNCTION WITH Mg^{2+}-Na^+-K^+-DEPENDENT ATP-ASE SYSTEM OF CELLS IN THE HUMAN FUNDIC GASTRIC MUCOSA. *(E.)* Mózsik, G.; Nagy, L.; Kutas, J.; Tárnok, F. (Univ. Med. Sch., Pecs, Hungary) *Scand. J. Gastroenterol.* 9(8): 741-745, 1974.

The Mg^{++}-Na^+-K^+-dependent ATP-ase system, which catalyzes the ATP into ADP, was biochemically prepared from human fundic gastric mucosa, and the effects of acetylcholine, carbamylcholine, neostigmine, and anticholinergics (atropine, isopropamide, and methyl-tropinium-bromide-xanthene-9-carboxylate) on this system were studied. A significant increase of total ATP-ase activity was caused by acetylcholine, carbamylcholine, and neostigmine. Acetylcholine caused an increase of Mg^{++}-dependent, total, and Na^+-K^+-dependent ATP-ase activity. Inhibition of total and Na^+-K^+-dependent activity was caused by atropine, while no inhibition was found on the Mg^{++}-dependent part alone. The inhibition of the transport ATP-ase by isopropamide and methyl-tropinium-bromide-xanthene-9-carboxylate were much less than that caused by atropine. Atropine prevented the effects of acetylcholine on the transport ATP-ase system. The stimulation or inhibition of this system by cholinergic or anticholinergic depended on the magnitudes of transport ATP-ase activity. This ATP-ase system may be a regulator of the effects of hormones and drugs in the human fundic gastric mucosa.

2006 MUCOUS CHANGE IN THE HUMAN DUODENUM: A LIGHT AND ELECTRON MICROSCOPIC STUDY AND CORRELATION WITH DISEASE AND GASTRIC ACID SECRETION. *(E.)* Patrick, W. J. A.; Denham, D.; Forrest, A. P. M. (Dep. Clin. Surg., Univ. Edinburgh, Scotland) *Gut* 15(10):767-776, 1974.

The significance of mucous change in the human duodenum in a series of patients with peptic ulcer disease was studied using light and electron microscopy. Mucosal biopsies from areas clear of any recognizable pathological change were taken from the 1st part of the duodenum in 117 patients suspected of having peptic ulcer disease. Light microscopy revealed mucous changes in 68 of the 117 biopsies and these changes were found only in patients with peptic ulcers (with 1 exception). Mucous change was more frequent when the acid levels were greater than 30 mEq/hr, but there was no specific relationship between the extent of mucous change and gastric acid secretion. Additional biopsies from the edge of active ulcers showed greater mucous change. There was an increased prevalence of epithelial damage, acute inflammatory cell reaction, and villous and crypt atrophy associated with mucous change. It was demonstrated under electron microscopy that there was a continuity between the ducts of hyperplastic Brunner's glands and crypts containing mucous change with a transition zone with epithelial cells with features of both Brunner's glands and mucous change. Thus mucous cells may arise by transformation of Brunner's gland cells or as a distinctive population in the crypts. The mucous change is probably a protective mechanism involved in some way with the healing of ulcers.

2007 INFLUENCE OF FEEDING AND SHAM FEEDING UPON SERUM GASTRIN AND GASTRIC ACID SECRETION IN CONTROL SUBJECTS AND DUODENAL ULCER PATIENTS. *(E.)* Mayer, G.; Arnold, R.; Feurle, G.; Fuchs, K.; Ketterer, H.; Track, N. S.; Creutzfeldt, W. (Dep. Med., Univ. Gottingen, Germany). *Scand. J. Gastroenterol.* 9(8):703-710, 1974.

The effect of feeding and a modified sham feeding procedure on gastric secretion and serum immunoreactive gastrin (IRG) concentrations in duodenal ulcer patients and in controls was studied. The investigations were performed in 19 controls, without known diseases of the gastrointestinal tract, and in 18 duodenal ulcer patients. Serum IRG increased significantly more, after a standard liquid meal (Vivasorb), in patients than in controls, while the fasting IRG levels did not significantly differ. A modifed sham feeding, where the subjects saw, tasted, chewed, and spat out an appetizing meal, stimulated gastric acid secretion in all subjects tested. The mean serum

IRG response of patients to modified sham feeding was significantly greater than the slight response of the controls. The increased IRG levels in patients could not be explained, and no correlation was found between the gastric acid response to modified sham feeding and the IRG output in patients and controls. The results suggest that acid secretion after vagal stimulation in man is mainly achieved by direct effects upon the parietal cells, and that vagally released IRG does not contribute significantly to the amount of acid secreted after vagal stimulation.

2008 INCREASED SENSITIVITY TO STIMULATION OF ACID SECRETION BY PENTAGASTRIN IN DUODENAL ULCER. *(E.)* Isenberg, J. I.; Grossman, M. I.; Maxwell, V.; Walsh, J. H. (VA Wadsworth Hosp. Cent., Los Angeles, Calif.). *J. Clin. Invest.*, 55(2):330-337, 1975.

Responsiveness of acid secretion to stimulation by pentagastrin was studied in 20 male duodenal ulcer patients and in 20 male patients without duodenal ulcer (controls). Dose-response studies were performed using graded doses of pentagastrin (2.7-6,000 ng/kg/hr). Differences in basal acid secretion between the 2 groups were not statistically significant. Each dose of pentagastrin (from 75-6,000 ng/kg/hr) produced significantly greater mean rates of acid secretion in the duodenal ulcer group than in the controls, and the highest observed response was greater in duodenal ulcer patients. The calculated maximal response (CMR) as determined by linear regression analysis, was greater in the duodenal ulcer group, while the dose for half maximal response was significantly less. By parallel line bioassay control subjects required 2.82 times more pentagastrin (95% confidence limits 2.12-3.74) than patients for equal response, expressed as percent of highest response. In neither group was there any significant correlation between sensitivity and basal acid output or highest observed response to pentagastrin. Thus duodenal ulcer patients not only secrete more acid in response to pentagastrin than patients without duodenal ulcer, a reflection of the greater parietal cell mass, but also are more sensitive to pentagastrin, requiring lower doses to achieve a certain fraction of maximal response. Several hypotheses were offered to explain this increased sensitivity, which may play a role in the pathogenesis of duodenal ulcer disease.

2009 INHIBITION OF Mg^{2+}-Na^{+}-K^{+}-DEPENDENT ATPase SYSTEM FROM HUMAN GASTRIC MUCOSA BY PROSTAGLANDINS E_1 AND E_2. *(E.)* Mozsik, G.; Kutas, J.; Nagy, L.; Nemeth, G. (Univ. Med. Sch., Pecs, Hungary). *Eur. J. Pharmacol.* 29(1):133-137, 1974.

2010 THE G CELL POPULATION OF THE GASTRIC ANTRUM, PLASMA GASTRIN, AND GASTRIC ACID SECRETION. *(E.)* Royston, C. M. S.; Polak, J. M.; Bloom, S.; Cooke, W. M.; Russell, C. R. C.; Pearse, A. G. E.; Baron, J. H.; Spencer, J.; Welbourn, R. B. (R. Postgrad. Med. Sch., London, England). *Gut* 15 (10):840-841, 1974.

2011 A COMPARISON OF BASAL AND STIMULATED GASTRIC AND PANCREATIC SECRETION IN PATIENTS WITH AND WITHOUT DUODENAL ULCER DISEASE. *(E.)* Gutierrez, L. V.; Baron, J. H. (R. Postgrad. Med. Sch., London, England). *Gut* 15(10):836, 1974.

2012 A DUODENOSCOPIC STUDY OF THE RELATIONSHIP BETWEEN DUODENAL ULCER DEPTH AND GASTRIC ACID OUTPUT. *(E.)* Moshal, M. G. (King Edward VIII Hosp., Durban, South Africa). *S. Afr. J. Surg.* 12 (2):68-69, 1974.

2013 GASTROINTESTINAL COMPLICATIONS OF STRESS. *(Fr.)* Sibilly, A.; Boutelier, P. (Munic. Hosp., Strasbourg, France). *J. Chir. (Paris)* 108 (1/2):117-134, 1974.

2014 CLINICAL AND PATHOPHYSIOLOGICAL ASPECTS OF GASTRODUODENAL LESIONS INDUCED BY CORTICOSTEROID THERAPY. *(Fr.)* Bleichner, G.; Mignon, F.; Mignon, M. (Bichat Hosp., Paris, France). *Ann. Med. Interne* 125(12):903-912, 1974.

2015 BLEEDING PEPTIC ULCERS IN WORKERS FROM THE HEAT-TREATING SECTIONS OF A METALLURGICAL PLANT. *(Rus.)* Shuliak, L. P.; Demchenko, P. S.; Polishchuk, Iu. P. (Zaporozhe Med. Inst., USSR). *Klin. Khir.* (9):76-78, 1974.

2016 EPIDEMIOLOGICAL STUDY ON PEPTIC ULCER AND GASTRIC CANCER IN THE CHINESE. *(E.)* Sung, J. L.; Wang, T. H.; Lu, T. H.; Wu, S. C.; Hsu, T. W.; Lee, T. H. (Natl. Taiwan Univ. Hosp., Taipei). *Rend. Gastroenterol.* 6(2):111-115, 1974.

2017 BENIGN GASTRIC ULCERS OCCURING WITH PERSISTENT HISTAMINE-FAST ACHLORHYDRIA. *(E.)* Wald, A.; Burbridge, E. J. (Johns Hopkins Univ. Sch. Med., Baltimore, Md.). *Hopkins Med. J.* 135(6):436-441, 1974.

2018 RELATIONS BETWEEN HYPERTHYROIDISM AND PEPTIC ULCER. *(Ger.)* Aoyagi, K.; Sengoku, K.; Hatano, M.; Hamaguchi, E.; Shimomura, T. (Med. Dent. Univ., Tokyo, Japan). *Dtsch. Z. Verdau. Stoffwechselkr.* 33(6):281-291, 1973.

2019 ANASTOMOTIC ULCERS. *(Rus.)* Podoprigora, A. P.; Nevmerzhitskii, V. M.; Miskov, A. P. (Shepetovka Cent. Reg. Hosp., Khmelnitska Reg., USSR). *Klin. Khir.* (9):71-72, 1974.

2020 A DOUBLE BLIND TRIAL OF CARBENOXOLONE AND GERANYL FARNESYLACETATE IN GASTRIC ULCER. *(E.)* Smith, P. M.; Sladen, G. E.; Langman, M. J. S.; Lennard-Jones, J. E. (Univ. Hosp. Wales, Cardiff). *Gut* 15(10):833, 1974.

2021 EFFECTIVE TREATMENT OF GASTRIC ULCERATION WITH A BISMUTH PREPARATION (DE-NOL). *(E.)* Boyes, B. E.; Woolf, I. L.; Wilson, R. Y.; Cowley, D. J.; Dymock, I. W. (Univ. Hosp. South Manchester, England). *Gut* 15(10):833, 1974.

2022 PROMISING THERAPY, USING A SULFOGLYCOPEPTIDE (SULGLYCOTIDE), FOR PEPTIC ULCER. *(It.)* Ruffa, S.; Ghirlanda, G.; Fedeli, G.; Greco, A. V. (Inst. Spec. Med. Pathol., Cathol. Univ. Sacred Heart, Rome, Italy). *Clin. Ter.* 68(5):469-476, 1974.

2023 RESULTS OBTAINED WITH DOGMAGEL IN THE TREATMENT OF GASTRIC AND DUODENAL ULCERS AND GASTRODUODENITIS. *(Sp.)* Sanchez Garcia-Moreno, I. (Sch. Gastrointest. Dis., I.N.P., Madrid, Spain). *Rev. Esp. Enferm. Apar. Dig.* 44(7):743-748, 1974.

2024 GASTRIC ULCER TREATED WITH SEGMENTAL RESECTION WITHOUT PYLOROPLASTY. *(E.)* Buhl, O.; Campbell, U. D. (Bispebjerg Hosp., Copenhagen, Denmark). *Acta Chir. Scand.* 140(7):528-530, 1974.

2025 OUR EXPERIENCE WITH OXYNTIC CELL VAGOTOMY (O.C.V.) IN THE TREATMENT OF DUODENAL ULCER. *(Sp.)* Dovale, M.; Vila, R.; Lozano, M.; Charlo, T. (Virgen del Rocio Munic. Hosp., Seville, Spain). *Rev. Esp. Enferm. Apar. Dig.* 44(7):651-676, 1974.

2026 OBSERVER VARIATION IN THE ASSESSMENT OF RESULTS OF SURGERY FOR PEPTIC ULCERATION. *(E.)* de Dombal, F. T.; Horrocks, J. C.; Clamp, S. E. (Gen. Infirm., Leeds, England). *Gut* 15(10):824, 1974.

2027 THE PATIENT'S ASSESSMENT OF THE RESULT OF OPERATION FOR PEPTIC ULCER. *(E.)* Cay, E. L.; Philip, A. E.; Small, W. P.; Hendersen, M. A.; Neilson, J. (West. Gen. Hosp., Edinburgh, Scotland). *Gut* 15(10):833-834, 1974.

2028 THE OPERATED STOMACH: CORRELATIONS BETWEEN ENDOSCOPIC AND RADIOLOGICAL FINDINGS. *(Ger.)* Realini, S.; Arnold, J.; Bugnion, M.; Fasel, J.; Fluckiger, A.; Hofstetter, J. R. (Med Polyclin., Univ. Lausanne, Switzerland). *Z. Gastroenterol.* 12(1):27-28, 1974.

2029 ENDOSCOPIC FINDINGS IN PATIENTS WITH OPERATED STOMACHS. *(Ger.)* Schmid, E.; Krautheim, J.; Blaich, E.; Oltmanns, D. (Reg. Hosp., Goppingen, Germany). *Z. Gastroenterol.* 12(1):29-30, 1974.

2030 THE ASSESSMENT OF FAILURES IN THE SURGICAL TREATMENT OF DUODENAL ULCER. *(Sp.)* Molins Benedetti, M.; Martinez Navarro, M.; Vila Planas, J. M. (no affil.). *An. Med. (Barc.)* 60(3):264-270, 1974.

2031 FOLLOW-UPS ON PATIENTS AFTER THE BILLROTH II PROCEDURE BY ENDOSCOPIC BIOPSIES AND ANALYSIS OF GASTRIC SECRETION. *(Ger.)* Schwamberger, K.; Reissigl, H.; Falser, N. (Surg. Clin., Univ. Innsbruck, Austria). *Z. Gastroenterol.* 12(2):113-116, 1974.

2032 SEQUELAE OF BILLROTH II GASTROENTEROSTOMY. *(Rus.)* Gevorkian, I. Kh.; Agadzhanian, G. I.; Balaian, R. A. (Erevan Med. Inst., USSR). *Klin. Khir.* (9):18-22, 1974.

2033 ROUX-EN-Y ANASTOMOSIS IN THE TREATMENT OF CHRONIC AFFERENT LOOP SYNDROME. *(E.)* Nielsen, O. V.; Jorgensen, S. P.; Pedersen, G. (Rigshosp., Univ. Copenhagen, Denmark). *Acta Chir. Scand.* 140(7):531-534, 1974.

2034 EARLY BILIOUS VOMITING AFTER GASTROENTEROSTOMY. *(E.)* Mathieson, A. J. M.; McDonald, C. W. E. (Bridge Earn Hosp., Perthshire, Scotland). *J. R. Coll. Surg. Edinb.* 19(4):216-221, 1974.

2035 EMPTYING OF THE SMALL INTESTINE BEFORE AND AFTER PEPTIC ULCER SURGERY. *(Rus.)* Siakerskii, A. G. (Irkutsk Med. Inst., USSR). *Klin. Khir.* (9):65-66, 1974.

2036 DISABILITY FOLLOWING GASTRECTOMY FOR PENETRATING GASTRIC AND DUODENAL ULCERS. *(Rus.)* Korolkova, L. N. (Chaliabinsk Med. Inst., USSR). *Klin. Khir.* (9):5-8, 1974.

2037 EVALUATION OF GASTRIC ACID SECRETION IN PATIENTS WITH DUODENAL ULCERS BEFORE AND AFTER VAGOTOMY AND PYLOROPLASTY. *(Rus.)* Satsukevich, V. N. (S. M. Kirov Mil. Med. Acad., Leningrad, USSR). *Klin. Khir.* (9):15-18, 1974.

2038 CHANGES IN CARBOHYDRATE METABOLISM PRODUCED BY TRUNCAL VAGOTOMY. *(Rus.)* Iarugskii, E. E. (Tashkent Med. Inst., USSR). *Klin. Khir.* (9):12-14, 1974.

2039 AN INTRA-OPERATIVE TEST FOR COMPLETENESS OF VAGOTOMY — I. THE GRASSI METHOD. *(E.)* van den Berg, W. P. J.; Bornman, P. C. (no affil.). *S. Afr. J. Surg.* 12(2):69-70, 1974.

See also, 1696, 1727, 1928, 2298, 2331, 2342, 2345, 2380.

2040 LYMPHOID HYPERPLASIA IN PATIENTS WITH DIARRHEA — MANIFESTATION OF IMMUNOLOGIC DISTURBANCES? *(Ger.)* Rosch, W.; Witzel, L.; Fuchs, H. F.; Fritsch, E. v. (Med. Clin. Polyclin., Univ. Erlangen-Nuremberg, Erlangen, Germany). *Leber Magen Darm* 3(1):29-34, 1973.

Immunological, histological, and therapeutic aspects of nodular lymphoid hyperplasia in diarrhea are presented in connection with 4 cases of obstinate and periodical diarrhea in 4 males (aged 19, 33, 34, and 30 yr). Lymphatic hyperplasia of the small intestine, IgA and IgM deficiency, and exudative enteropathy were diagnosed in a 19-yr-old patient with predisposition to diarrhea since infancy. Gluten-sensitive enteropathy, hypogammaglobulinemia, and lymphatic hyperplasia of the small intestine were found in another patient with diarrhea persisting for 2 yr. Agamma-globulinemia, subtotal atrophy of the villi of the small intestinal mucosa, and suspect follicular hyperplasia were diagnosed in another case persisting for 2 yr. A fourth patient showed giardiasis and follicular hyperplasia. Nodular lymphoid hyperplasia of the small intestine was characterized radiographically by numerous small filling defects which were uniform in size and contour. Hypogammaglobulinemia, agammaglobulinemia, gluten-sensitive enteropathy, giardiasis, as well as IgA and IgM deficiency are characteristic findings of diarrhea with lymphoid hyperplasia. Gluten free diet in isolated IgA deficiency, and antibiotics and gammaglobulin in agammaglobulinemia are indicated for therapy.

2041 THE PATHOLOGICAL PHYSIOLOGY OF SMALL INTESTINE. *(E.)* Masuda, M. (Kyoto Prefect. Univ. Med., Japan). *Jpn. J. Med.* 13(3):165-171, 1974.

2042 LIPOHYPERPLASIA OF THE ILEOCAECAL REGION. *(E.)* Axelsson, C.; Andersen, J. A. (Sundby Hosp., Copenhagen, Denmark). *Acta Chir. Scand.* 140(8):649-654, 1974.

2043 RARE CASES OF CONGENITAL SHORT BOWEL. *(Ger.)* Dumke, K.; Schnoy, N. (Westend Clin., Free Univ., Berlin, Germany). *Z. Gastroenterol* 12(5):321-325, 1974.

2044 MEMBRANE DIGESTION IN THE FUNCTIONAL DIAGNOSIS OF THE SMALL INTESTINE. *(Rus.)* Zlatkina, A. R.; Misautova, A. A.; Makievskaia, S. E.; Kutychkina, O. A. (M. F. Vladimirskii Reg. Clin. Res. Inst., Moscow, USSR). *Klin. Med. (Mosk.)* 52(11):106-110, 1974.

2045 ERRORS IN THE DIAGNOSIS AND TREATMENT OF CLOSED INTESTINAL TRAUMA. *(Rus.)* Rarenko, A. S. (Krasnodon Cent. Munic. Hosp., USSR). *Klin. Khir.* (8):36-38, 1974.

2046 INTRAABDOMINAL ISCHEMIA. *(Ger.)* Baas, E. U. (1st Med. Clin., Johannes Gutenberg Univ., Mainz, Germany). *Therapiewoche* 22(24):2513-2522, 1974.

2047 INFLAMMATORY STENOSIS OF THE SMALL AND LARGE INTESTINES. *(Ger.)* Schier, J.; Pfriender, H.; Strube, H. D. (Surg. Clin., Univ. Mainz, Germany). *Therapiewoche* 24(18):2049-2055, 1974.

2048 LATE RADIATION DAMAGE IN THE SMALL AND LARGE INTESTINE. *(Ger.)* Lenner, V. (Surg. Clin., Univ. Mainz, Germany). *Therapiewoche* 24(18):2055-2061, 1974.

2049 THE VALUE OF ENDOSCOPIC EXAMINATION IN THE DIAGNOSIS OF DUODENITIS. *(Ger.)* Cheli, R.; Aste, H.; Ciancamerla, G. (San Martino Hosp., Genoa, Italy). *Z. Gastroenterol.* 12(1):41, 1974.

2050 EROSIVE DUODENITIS. *(Rus.)* Smagin, V. G.; Sokolov, L. K.; Gavrilenko, Ia. V.; Zotov, V. M.; Ivanov, V. V. (4th Dep. Ministry Health, Moscow, USSR). *Klin. Med. (Mosk.)* 52(7):93-94, 1974.

2051 SMALL INTESTINE PERFORATION AS A COMPLICATION OF TYPHOID FEVER. *(Ser.)* Tomanovic, A.; Baljosevic, S.; Moracic, B.; Gigic, A.; Ristic, T.; Slavkovic, G. (Gen. Hosp., Pristina, Yugoslavia). *Vojnosanit. Pregl.* 31(4):239-241, 1974.

2052 THE SCINTIGRAPHIC DETECTION OF THE DIVERTICULUM OF MECKEL: A RARE BUT INTERESTING ISOTOPIC EXPLORATION PROCEDURE IN GASTRO-ENTEROLOGY. *(E.)* De Somer, E.; Boeckx, L.; De Roo, M. J. K.; Kerremans, R.; Frijns, J. P. (St. Rafael Acad. Hosp., Cathol. Univ., Louvain, Belgium). *J. Belge Radiol.* 57(3):179-184, 1974.

2053 DUODENAL DIVERTICULUM WITH INTERMITTENT BILIARY STASIS. *(E.)* Solhaug, J. H.; Semb, B. K. H. (Dep. Surg., Univ. Bergen, Norway). *Acta Chir. Scand.* 140(8):670-673, 1974.

2054 SURGICAL TREATMENT OF A DUODENAL DIVERTICULUM LOCATED INSIDE THE PANCREAS. *(Rus.)* Mikhailichenko, A. F. (Cent. Reg. Hosp., Crimean Reg., USSR). *Klin. Khir.* (9):75, 1974.

2055 EARLY POSTOPERATIVE INTESTINAL OBSTRUCTION DUE TO ADHESIONS. *(Rus.)* Toropov, Iu. D. (M. Gorky Inst. Postgrad. Med., Zaporozhe, USSR). *Klin. Khir.* (8):22-26, 1974.

2056 THE SITE AT WHICH THE SMALL INTESTINE IS RESECTED IN ACUTE OBSTRUCTION. *(Rus.)* Kolibaba, S. S. (N. I. Pirogov Med. Inst., Vinnitsa, USSR). *Klin. Khir.* (8):30-33, 1974.

2057 RARE FORM OF INTESTINAL OBSTRUCTION. *(Rus.)* Seniutovich, V. F.; Shevchuk, M. G.; Ostapenko, E. A. (Ivano-Frankovsk Med. Inst., USSR). *Klin. Khir.* (8):58-59, 1974.

2058 DOUBLE PRIMARY CANCERS OF THE SMALL INTESTINE AND THE STOMACH: REPORT OF A CASE. *(E.)* Matsuo, T.; Yao, T.; Miyasaki, K. (Hyogo Prefect. Awaji Hosp., Sumoto, Japan). *Kobe J. Med. Sci.* 20(3/4)97-102, 1974.

2059 INTUSSUSCEPTION CAUSED BY A LYMPHOMA IN CHILDHOOD: ONE CASE IN AN EIGHT-YEAR-OLD BOY. *(Sp.)* Vilaseca, J.; Garnacho, A.; Llorens, V.; Blanes, R.; Bonafonte, M.; Mari, L. (Munic. Social Welfare Hosp., Barcelona, Spain). *Rev. Esp. Enferm. Apar. Dig.* 44(6):605-612, 1974.

2060 RETICULUM CELL SARCOMATOSIS OF THE SMALL INTESTINE. *(Rus.)* Bakhtiiarov, V. A. (Kemerovo Med. Inst., USSR). *Klin. Khir.* (10):44-45, 1974.

2061 CYSTIC LYMPHANGIOMA OF THE MESENTERY OF THE SMALL INTESTINE IN A CHILD. *(Rus.)* Stetskiv, O. V. (Ichnia Cent. Reg. Hosp., Chernigov Reg., USSR). *Klin. Khir.* (11):85-86, 1974.

2062 LYMPHOSARCOMA OF THE SMALL INTESTINE. *(Rus.)* Podoprigora, A. P. (Shepetovka Cent. Reg. Hosp., USSR). *Khirurgiia (Mosk.)* (3): 114-116, 1973.

2063 PROLONGED SURVIVAL IN A PATIENT WITH LYMPHOSARCOMA OF THE SMALL INTESTINE TREATED WITH COMBINED THERAPY. *(Rus.)* Budarina, E. M.; Kopevalov, R. V. (S. M. Kirov Med. Inst., Gorki, USSR). *Klin. Khir.* (10):46-47, 1974.

2064 CLINICAL SURVEY OF THE PATIENTS WITH MASSIVE RESECTION OF SMALL INTESTINE DURING THE LAST TEN YEARS. *(E.)* Chen, K. M. (Coll. Med., Natl. Taiwan Univ. Taipei). *J. Formosan Med. Assoc.* 73(1):35-44, 1974.

2065 DIET AND GROWTH AFTER RESECTION OF ILEUM IN CHILDHOOD. *(E.)* Valman, H. B. (Northwick Park Hosp., Harrow, England). *Gut* 15(10):822, 1974.

2066 DECREASE OF CALCIUM-BINDING PROTEIN IN THE SMALL INTESTINAL MUCOSA IN UREMIC PATIENTS WITH RENAL OSTEODYSTROPHY. *(Ger.)* Piazolo, P. J.; Hotz, J.; Helmke, K.; Franz, H. E.; Goebell, H. (Cent. Intern. Med. Pediatr., Univ. Ulm, Germany). *Z. Gastroenterol.* 12(4):273-276, 1974.

See also, 1675, 1885, 1891, 1892, 1898, 1899, 1936, 1957, 2023, 2127, 2147, 2294, 2325, 2366, 2389, 2394.

SMALL INTESTINE

Malabsorption

2067 THE SMALL-INTESTINAL MUCOSA IN COW'S MILK ALLERGY. *(E.)* Shiner, M.; Ballard, J.; Smith, M. E. (Cent. Middlesex Hosp., London, England). *Lancet* 1(7899):136-140, 1975.

Two infants (1 boy and 1 girl, aged 6-8 weeks) were investigated for allergy to cow's milk protein. The initial biopsies of both patients seemed normal, but subsequent biopsies showed the presence of shortened, broader villi which were in part convoluted. In the male patient, before receiving milk, the epithelium microvilli, lysosomes, mitochondria and golgi were normal. The sub-epithelium basement membrane and connective tissue were thickened. The appearance of the inflammatory cells was normal for mast cells and plasma cell activity; eosinophils and polymorphs were absent. After receiving milk, edema in the connective tissue, and an increase in the plasma cell activity were noted in the sub-epithelium. For the infant girl, the initial biopsy differed from the boy only in that the sub-epithelium basement membrane appeared normal with some edema noted in connective tissue before milk was given. After milk was given, the biopsy showed by electron microscopy that in the epithelium, the microvilli were irregular, lysosomes increased, mitochondria were damaged, and golgi were active; for the sub-epithelium connective tissues patchy edema and coarsening of fibers (collagen?) were noted; and the mast cells, plasma cell activity, eosinophils and polymorphs were increased with the mast cells appearing degranulated. Plasma-cells producing IgG, IgM, and IgE were slightly increased in the boy's 1st biopsy. After milk there was a further increase in IgM and IgE plasma-cells, but little change in IgA and none in IgG cells or in complement. Basement membrane and connective tissue fluorescence were noted with IgG, IgE, and IgA antisera. The initial biopsy specimen from the infant girl showed a normal staining pattern of plasma-cells, but there was patchy membrane staining with antisera to IgE and C3. A striking increase in mucosal IgM and IgE plasma-cells associated with staining of the epithelial brush borders and inter-epithelial spaces 11 1/2 hr after milk suggests that these immunoglobulins are secreted into the bowel lumen. Connective-tissue staining with antisera to IgG and C3 was also in-

creased, but no change in IgG or IgA plasma-cells was noted. These studies indicate that, after ingestion of cow's milk by infants allergic to milk proteins, reaginic immune reactions occur in the small intestinal mucosa. These findings may provide a sound basis for diagnosis of such an allergy and for the treatment of similar patients with disodium cromoglycate.

2068 IDENTIFYING TOXIC FRACTIONS OF WHEAT GLUTEN AND THEIR EFFECT ON THE JEJUNAL MUCOSA IN COELIAC DISEASE. *(E.)* Dissanayake, A. S.; Jerrome, D. W.; Offord, R. E.; Truelove, S. C.; Whitehead, R. (Radcliffe Infirm., Oxford, England). *Gut* 15(12):931-946, 1974.

The toxicity of 3 fractions (A, B, and C) obtained by ultrafiltration of a peptic:tryptic digest of gluten was assessed by serial feeding experiments in 3 patients with treated celiac disease. One patient evidenced no clinical features of a reaction to the feeding. A second patient began having symptoms about the 4th day and these persisted over 3 weeks after the challenge. The symptoms were mild and consisted of malaise, occasional upper abdominal discomfort, and mild diarrhea. The third patient noted symptoms about 5 hr after a large single dose of fraction B. Generalized abdominal discomfort, distention and vomiting occurred. All symptoms passed by 24 hr. No consistent changes occurred in any patient in routine blood investigations. The first fraction, A, contained amino acids and oligopeptides and produced no damage to the jejunal mucosa. The other 2 fractions both caused mucosal damage. Fraction B contained the products of digestion of smaller molecular weight, polypeptides in the region of 8000 molecular weight. Thus it contained no gliadin or gluten. Ultrastructural evidence of damage was visible 6 hr after challenge with fraction B, and by 10 hr histological abnormalities were also present. Ultrastructural abnormalities occurred early in the epithelial cells and preceded changes in the basement membrane and capillaries. In all 3 patients the disaccharidases in the initial biopsy were within normal limits. In one patient no change in enzyme content was noted at 6 hr, but by 24 hr, a precipitous fall had occurred with all 3 enzymes which continued to fall over the next 2 days. In a second patient the jejunal disaccharidases were reduced at 10 hr and fell further by 24 hr. In a third patient a similar but more marked fall occurred after 9 hr with a single large dose of fraction B. Enzymes were lower at 24 hr, but by 72 hr there was evidence of enzyme recovery. It is concluded from this study that the rapid onset of damage after challenge, coupled with the evidence of recovery as early as 72 hr later, is more in keeping with a direct action on the surface epithelial cells rather than an immune mechanism.

2069 ULTRASTRUCTURAL FEATURES OF ACRODERMATITIS ENTEROPATHICA. *(Ger.)* Lombeck, I.; von Bassewitz, D. B. (Pediatr. Clin., Univ. Dusseldorf, Germany). *Monatschr. Kinderheilk.* 122(7):661-662, 1974.

Electron microscopic examinations were performed on biopsies from the small intestine of a 13-month-old boy with acrodermatitis enteropathica. Biopsies were taken before institution of therapy and 6 weeks and 7 and 12 months thereafter. Therapy consisted of unboiled breast milk combined, 4 weeks later, with iodochlorhydroxyquin (750 mg/day). Before therapy, atypical inclusions were found in Paneth's cells. These inclusions were oval or rhomboid and, in most cases, larger than the acidophilic granules. They consisted of electron-dense material surrounded by less dense material with a fibrous structure. The rest of the intestinal epithelium appeared normal except for the absence of some microvilli and infiltration of the lamina propria by granulocytes, lymphocytes, eosinophils, macrophages, and mast cells. Although clinical improvement occurred and inflammatory changes in the small intestine regressed with therapy, the inclusions in Paneth's cells persisted in the next 3 biopsies. Examinations performed under the light microscope disclosed regeneration of the villi after 6 months of therapy and complete normalization of the villi after 12 months. When the boy was 21 months old, a normal diet was gradually introduced. However, drug treatment is being continued since the child developed a recurrence of symptoms when iodochlorhydroxyquin was discontinued at age 30 months.

2070 MEGADUODENUM. A CASE WITH BACTERIAL OVERGROWTH, DECONJUGATION OF BILE SALTS, MALABSORPTION, AND POLYNEUROPATHY. *(E.)* Matzen, P.; Davidsen, D.; Mathiasen, M. S. (Marselisborg Hosp., Aarhus, Denmark). *Scand. J. Gastroenterol.* 9(7): 645-650, 1974.

A case history is reported of a 50-yr-old man suffering from severe duodenal dilatation and stasis proximal to the crossing of the intestine by the superior mesenteric artery, as well as from malabsorption. For 6 months prior to hospital admission the patient had 3-4 voluminous, gray, fatty stools/day and a weight loss of about 10 kg. In the last few weeks before admission appetite decrease was accompanied by increasing epigastric pain and voluminous vomiting. On admission the abdomen was distended with a tender doughy mass in the epigastrium. Roentgenographic examination of the stomach and duodenum and hypotonic duodenography in combination with angiography of the superior mesenteric artery revealed an extremely dilated duodenum with retention orally to an impression in the horizontal part of the duodenum, in which the superior mesenteric artery was demonstrated. On aerobic culture of the duodenal aspirate, 10^6 colonies *Klebsiella pneumoniae*/ml and 5 x 10^4 colonies nonhemolytic streptococci were found. On breath test, where glycine-1-^{14}C-cholic acid was administered p.o., 0.41% ^{14}C was recovered in the expiratory air carbon dioxide (normal < 0.10%), whereas 2% ^{14}C was excreted in the feces (normal < 7%), indicating premature deconjugation of bile salts in the upper small intestine, but intact reabsorption of bile salts in the terminal ileum. During the course of the condition, polyneuropathy had developed which could not be explained by folic acid, B_{12}, or thiamine deficiency but was possibly due to deficiency of another essential nutrient fac-

tor. The patient was treated with nasogastric suction and parenteral fluid, electrolytes, and nutrition but his condition deteriorated. A duodenojejunostomy was performed but the patient died postoperatively from mesenteric thrombosis. Although long-term treatment with antibiotics has been suggested in other publications, the authors prefer operative treatment in cases of megaduodenum with malabsorption due to abnormal bacterial flora. Despite the outcome of this case, it is suggested that duodenojejunostomy is the operation of choice.

2071 THE HL-A SYSTEM AND THE IMMUNOLOGICAL RESPONSE TO DIETARY ANTIGENS. *(E.)* Scott, B. B.; Swinburne, L. M.; Rajah, S. M.; Losowsky, M. S. (St. James's Univ. Hosp., Leeds, England). *Gut* 15(10):823, 1974.

2072 TISSUE DAMAGE MEDIATED BY LYMPHOCYTES IN LOCAL SMALL INTESTINAL IMMUNE REACTIONS. *(E.)* Ferguson, A.; McDonald, T. T. (Dep. Bacteriol. Immunol., Univ. Glasgow, Scotland). *Gut* 15(10):834, 1974.

2073 CLINICAL SIGNIFICANCE OF DISORDERS IN DISACCHARIDE HYDROLYSIS AND ABSORPTION IN THE SMALL INTESTINE. *(Rus.)* Levin, A. A. (Leningrad Med. Inst. Sanit. Hyg., USSR). *Klin. Med. (Mosk.)* 52(8):33-36, 1974.

2074 GASTROJEJUNOCOLONIC FISTULA, BLIND-LOOP SYNDROME, AND STEATORRHEA. *(Sp.)* de la Santa Lopez, J.; Velloso Jimenez, A.; Hernandez Pascual, A.; Belda Laguna, O. (Virgin del Rocio Munic. Hosp., Seville, Spain). *Rev. Esp. Enferm. Apar. Dig.* 44(5):493-508, 1974.

2075 MALABSORPTION SYNDROME. *(Ger.)* Ewe, K. (1st Med. Clin., Johannes Gutenberg Univ., Mainz, Germany). *Therapiewoche* 24(22):2522-2534, 1974.

2076 HYPOGAMMAGLOBULINEMIA IN PATIENTS WITH MALABSORPTION SYNDROME. *(Rus.)* Kuvaeva, I. B.; Vinogradova, M. A.; Temkina, T. Ia.; Kamaeva, N. G.; Makeeva, A. A. (All-Union Res. Inst. Gastroenterol., Moscow, USSR). *Klin. Med. (Mosk.)* 52(8):124-131, 1974.

2077 MALABSORPTION IN AFRICA. *(E.)* Cook, G. C. (Dep. Med., Univ. Zambia, Lusaka). *Trans. R. Soc. Trop. Med. Hyg.* 68(6):419-435, 1974.

2078 LACTOSE INTOLERANCE CAUSED BY LACTASE DEFICIENCY. *(Fr.)* Duchier, J. (Hotel Dieu, Paris, France). *Arch. Fr. Mal. App. Dig.* 63(6):513-531, 1974.

2079 THE INHERITANCE OF SELECTIVE ADULT-TYPE LACTOSE MALABSORPTION. *(E.)* Sahi, T. (Dep. Public Health Sci., Univ. Helsinki, Finland). *Scand. J. Gastroenterol.* [*Suppl.*] 9(30):73 pp., 1974.

2080 LACTOSE MALABSORPTION IN CHILDREN WITH CHRONIC NONSPECIFIC DIARRHEA (IRRITABLE COLON). *(Sw.)* Norstedt, S. (no affil.). *Lakartidningen* 71(21):2181-2182, 1974.

2081 DRUG-INDUCED MALABSORPTION OF VITAMIN B_{12}. VII. MALABSORPTION OF B_{12} DURING TREATMENT WITH POTASSIUM CITRATE. *(E.)* Palva, I. P.; Salokannel, S. J.; Palva, H. L. A.; Rytokonen, U.; Timonen, T. (Dep. Med., Univ. Kuopio, Finland). *Acta Med. Scand.* 196(6):525-526, 1974.

2082 INTESTINAL IRON ABSORPTION UNDER THE INFLUENCE OF AVAILABLE STORAGE IRON AND ERYTHROBLASTIC HYPERPLASIA. COMPARATIVE STUDIES IN CHILDREN WITH HEREDITARY SPHEROCYTOSIS, NONSPHEROCYTIC ENZYMOPENIC HEMOLYTIC ANEMIA, ACQUIRED HEMOLYTIC ANEMIA, VITAMIN B_{12} DEFICIENCY INDUCED MEGALOBLASTIC ANEMIA, ERYTHROBLASTIC HYPERPLASIA AND APLASTIC ANEMIA. *(E.)* Bender-Gotze, C.; Heinrich, H. C.; Gabbe, E. E.; Oppitz, K. H.; Schafer, K. H.; Schroter, W.; Whang, D. H. (Hamburg-Eppendorf Univ. Hosp., Hamburg, Germany). *Z. Kinderheilkd.* 118(4):283-301, 1975.

2083 TROPICAL SPRUE IN RHODESIA. *(E.)* Thomas, G. E.; Clain, D. J. (Dep. Med., Univ. Rhodesia, Salisbury). *Gut* 15(10):823, 1974.

2084 HISTOCOMPATIBILITY ANTIGENS IN COELIAC DISEASE. *(E.)* Ferguson, R.; Asquith, P.; Cooke, W. T. (Gen. Hosp., Birmingham, England). *Gut* 15(10):834, 1974.

2085 FAMILY STUDY OF COELIAC DISEASE. *(E.)* Rolles, C. J.; Kyaw Myint, T. O.; Sin, W. K.; Anderson, C. M. (Inst. Child Health, Univ. Birmingham, England). *Gut* 15(10):827, 1974.

2086 THE PREVALENCE OF COELIAC DISEASE AND CYSTIC FIBROSIS IN IRELAND, SCOTLAND, AND ENGLAND AND WALES. *(E.)* O'Reilly, D.; Murphy, J.; McLaughlin, J.; Bradshaw, J.; Dean, G. (Dep. Med., Univ. Coll., Cork, Ireland). *Int. J. Epidemiol.* 3(3):247-251, 1974.

2087 EVIDENCE OF IMMUNODEFICIENCY IN PATIENTS WITH COELIAC DISEASE. *(E.)* Baker, P. G.; Jones, J. V.; Peacock, D. B.; Read, A. E. (Dep. Med., Univ. Bristol, England). *Gut* 15(10):835, 1974.

2088 ENTERIC LOSS OF LYMPHOCYTES IN COELIAC DISEASE AND IN CROHN'S DISEASE. *(E.)* Weetman, A. P.; Haggith, J.; Douglas, A. P. (R. Victoria Infirm., Newcastle upon Tyne, England). *Gut* 15(10):823, 1974.

2089 CONTINUING GLUTEN INGESTION AND ITS DETECTION IN TREATED COELIAC PATIENTS. *(E.)* Baker, P. G.; Read, A. E. (Bristol R. Infirm., England). *Gut* 15(10):827, 1974.

2090 LIGHT AND ELECTRON MICROSCOPE STUDIES OF SMALL INTESTINE BIOPSIES IN ACRODERMATITIS ENTEROPATHICA. *(Ger.)* Heilmann, K.; Rossner, J. A.; Braun, O. (Munic. Hosp., Pforzheim, Germany). *Verh. Dtsch. Ges. Pathol.* 57:450, 1973.

See also, 1834, 2001, 2252, 2349.

LARGE INTESTINE

2091 EFFECT OF DIET ON HUMAN FECAL FLORA: COMPARISON OF JAPANESE AND AMERICAN DIETS. *(E.)* Finegold, S.; Attebery, H. R.; Sutter, V. L. (VA Wadsworth Hosp. Cent., Los Angeles, Calif.). *Am. J. Clin. Nutr.* 27(12):1456-1469, 1974.

The composition of fecal flora was compared in 18 Japanese Americans living on a Western diet and 15 living on a traditional Japanese diet. The 2 groups were comparable in sex distribution and number of daily bowel movements, but there was a statistically significant age difference, those on the traditional diet being an average of 19.0 yr older. Significant differences were found in the following species of intestinal flora (figures represent mean count $\log_{10}$ number of organisms/g feces in Japanese diet and Western diet, resp.): *Eubacterium contortum* (9.58, 0), *E. lentum* (10.20, 10.07), *Bifidobacterium infantis* and other species (0, 10.29), *Peptostreptococcus species 1* (10.53, 0), *Peptostreptococcus species 1-25* (8.29, 4.64), *Streptococcus faecalis faecalis* (9.83, 8.46), and other facultative or aerobic organisms (7.20, 4.75). No statistically significant differences were found with *Clostridium paraputrificum* or other clostridia believed to modify bile acids into carcinogens. In all, 220 distinct species, subspecies, and groups were recovered from the subjects on the Japanese diet, and over 160 from those on the Western diet. It is concluded that diet may play a significant role in determining fecal floral composition, and that further study is indicated.

2092 CHROMOSOME BANDING PATTERN IN HUMAN COLONIC POLYPS. *(E.)* Mitelman, F.; Mark, J.; Nilsson, P. G.; Dencker, H.; Norryd, C.; Tranberg, K. G. (Univ. Hosp., Lund, Sweden). *Hereditas* 78(1): 63-68, 1974.

Chromosomal studies were compared between polyps from a patient with familial polyposis and sporadic adenomas from different patients with no familial polyposis. Five polyps were taken from the patient with the familial history and 4 from the sporadic cases. All were about 5 mm in diameter and were subjected to histological and chromosomal analysis. In the sporadic polyps, the normal diploid complement was predominant. In 2, no deviations in chromosome number were found; in 1, 1 hypodiploid cell was found; and in another, 5 of 23 cells had abnormal chromosome numbers. In contrast, all of the cells from the 5 polyps of the familial patient had deviations. In 3, deviant karyotypes were predominant, and in 1, no normal cells were found. G-band analysis showed that of 26 cells with a deviating karyotype all showed engagement of 1 of the C (chromosome 8 in 7 cells) and/or D (chromosome 14 in 8 cells) group chromosomes. Histologically, all the polyps were benign adenomas showing no sign of invasiveness. It is concluded that chromosomal changes occur before histologic evidence of invasiveness in the oncogenic process.

2093 THE OCCULT OSTEOMATOUS CHANGES IN THE MANDIBLE IN PATIENTS WITH FAMILIAL POLYPOSIS COLI. *(E.)* Utsunomiya, J.; Nakamura, T. (Tokyo Med. Dent. Univ., Japan). *Br. J. Surg.* 62(1):45-51, 1975.

Radiological examinations were made of the oral region in patients with familial polyposis coli (FPC) to determine the possible association of FPC with osteoma in the mandible. Routine medical examination of all the cases was done, with emphasis on radiology of the gastrointestinal tract, fiberscopy of the stomach and colon, palpation and inspection of the body surface, systemic bone survey and anamnesis of tumor removal, plus routine dental and oral radiological examinations. Radio-opaque lesions (characteristic of endosteoma) were detected in the mandibular bone in 27 of 29 cases in 15 families with FPC (21 cases without Gardner's stigmata, 7 cases with the incomplete syndrome, 1 case with the complete syndrome); 35.7% of the cases also showed impacted teeth. The occult (thought to correspond to central osteoma or endosteoma) osteomatous changes described are considered a useful finding for early detection of the polyposis carrier in the FPC family and provide further understanding of Gardner's syndrome; they also indicate that FPC has the potential to produce a variety of neoplastic lesions other than the colonic lesion.

2094 OCCURRENCE OF ATYPICAL CHANGES IN COLORECTAL POLYPS ACCORDING TO THE FAMILIAL HISTORY OF GASTRO-INTESTINAL TRACT MALIGNANCIES. *(E.)* Crespi, M.; Bigotti, A.; Di Matteo, S.; Casale, V. (Regina Elena Inst. Cancer Res., Rome, Italy). *Proc. Fifth Int. Symp. Biol. Characterization Hum. Tumors, Bologna, April 4-6, 1973* 1:269-275, 1974.

Nineteen hundred forty eight cases were selected out of 19,000 individuals for proctocoloscopy on the basis of 3 parameters: suspicious digital examination, occult blood in the stools, or suspicious personal or

family history. An 8.8% incidence of polypoid lesions was found, 24.6% being greater than 1 cm in diameter and 75.4% being smaller. Thirteen adenocarcinomas were found, all in Duke stage A. Presence of occult blood in the stools was found to be an invalid criterion, due to false positives from hemorrhoidal involvement. Digital examination was able to detect growths as small as 3 mm in diameter. The incidence of atypical changes in adenomatous polyps was significantly higher in patients with suspicious family histories. Of 74 cases of adenoma, a 58% incidence of atypical changes were found in polyps under 1 cm in diameter in contrast to a 14% incidence in controls; in polyps over 1 cm a 66% incidence was found compared with 25% in controls. No definite conclusions are drawn, but digital examination, and especially family history may prove valuable as screening criteria.

2095 THE ANATOMIC PRECURSOR OF COLORECTAL CARCINOMA. *(E.)* Fenoglio, C. M.; Lane, N. (Presbyt. Hosp., New York, N.Y.). *JAMA* 231(6):640-642, 1975.

Colorectal polyp anatomy and malignant potential is briefly reviewed. Polyps are defined as any visible protrusion on the colorectal mucous membrane. Hyperplastic polyps account for about 90% of all polyps and appear as minute (1-5 mm) dewdrop elevations. They result from increase in cell division in the lower parts of the crypts of Lieberkuhn, and are not neoplastic. Adenomas, in contrast, are benign neoplasms, and occur in 2 major forms, pedunculated and sessile, although they can occur in all shapes and sizes. They represent a serious disturbance of cellular renewal in which unrestricted mitosis takes place at all tissue levels and does not result in complete differentiation. Carcinomas tend to arise in pedunculated adenomas over 1.5 cm in diameter (10% frequency) and in sessile papillary or villous adenomas over 5 cm in diameter (30% frequency). These are only clinically significant when they become invasive (i.e. they penetrate the muscularis mucosae). Of every 1000 polyps, 900 are hyperplastic, 90 are under 1.5 cm, and of the remaining 10, 1 will have an invasive carcinoma. Evidence indicates that carcinomas do not arise *de novo* from normal mucosa without an adenomatous stage.

2096 RECTAL CARCINOMA: A TWO PART STUDY OF TUMOUR PROLIFERATION AND HOST CELLULAR IMMUNITY. *(E.)* Bone, G.; Camplejohn, R. S. (R. Victoria Infirm., Newcastle upon Tyne, England). *Am. J. Proctol.* 26(1):65-76, 1975.

Tumor proliferation and host cellular immunity were studied in patients with rectal adenocarcinoma. In a proliferation study, 19 patients were given the stathmokinetic drug vincristine sulfate (0.045 mg/kg). Biopsies of both normal mucosa and tumor were fixed, stained and counted for cells in mitosis. Cell cycle time for tumors was calculated at 192 hr, metaphase time at 2.0 hr, and total mitotic time at 2.3 hr. Comparison with normal mucosa showed that tumors had a lower fraction of the cell population proliferating, and exhibited a reduction in cell loss from 100% (normal) to 98.7%. An immunity study investigated the relation between host immunity and tumor proliferation and spread. Twenty-one of 40 patients were given proliferation studies, and were divided on a histological basis into well-differentiated, moderately well-differentiated, and poorly differentiated tumors. The patients were given skin tests for sensitivity to 2,4-dinitro-1-chlorobenzene and divided into groups sensitive to only 200 µg, to 100 µg, to 40 µg, or to 2 µg. Pathological grading of the patients was also done according to Duke's classification of degree of tumor spread. A significant positive correlation was found between competence in response to 2,4-dinitro-1-chlorobenzene and decreasing spread of the tumor. No correlation was found between proliferation rates and competent cellular immunity. It is concluded that cell-mediated immunity has no effect on proliferation of rectal carcinomas, since they proliferate by decreased cell loss, but there is an inverse relationship between tumor spread and host immunity.

2097 PROGNOSTIC EVALUATION OF REGIONAL LYMPH NODE MORPHOLOGY IN COLORECTAL CANCER. *(E.)* Tsakraklides, V.; Wanebo, H. J.; Sternberg, S. S.; Stearns, M.; Good, R. A. (Mem. Sloan-Kettering Cancer Cent., New York, N.Y.). *Am. J. Surg.* 129(2): 174-180, 1975.

Histological patterns of regional lymph nodes were studied with relation to prognosis in 143 patients with left-sided colorectal adenocarcinoma. The degree of infiltration of the tumor was classified into 3 stages, and the lymph node histologic patterns into 4 types: lymphocyte predominance, germinal center predominance, lymphocyte depletion, and unstimulated. Of these types, 20% had lymphocyte predominance, 41% had germinal center predominance, none had lymphocyte depletion, and 39% had unstimulated. Five yr survival rates were 71% for germinal center predominance, and 54% for lymphocyte predominance, and unstimulated, the difference not statistically significant. In contrast, survival rates were 82%, 62%, and 47%, resp., among the 3 groups classified according to the stage of tumor infiltration. It is concluded that in cancer of the colon, prognosis is not correlated with regional lymph node morphology, but with extent of the disease.

2098 ROLE OF SERIAL PLASMA C.E.A. ASSAYS IN DETECTION OF RECURRENT AND METASTATIC COLORECTAL CARCINOMAS. *(E.)* Mackay, A. M.; Patel, S.; Carter, S.; Stevens, U.; Laurence, D. J. R.; Cooper, E. H.; Neville, A. M. (R. Cancer Hosp., London, England). *Br. Med. J.* 4(5941):382-385, 1974.

Serial estimations of plasma carcinoembryonic antigen (CEA) levels have been carried out in 220 patients with colorectal carcinomas who had potentially and apparently curative resections. Of these patients, 167 were considered well and without clinical evidence of recurrent disease. In 150 of the 167 patients the plasma CEA levels remained in the normal

range throughout the follow-up period. In the remaining 17, progressively rising titers exceeding 40 ng/ml were observed. In the remaining 53 of the 220 patients, either recurrent or metastatic tumors or both developed during the assessment period. In 26 of these, clinical detection of recurrent tumors was preceded by plasma CEA titers rising to pathological levels. In a further 10, rising titers were noted synchronously with the clinical detection of the tumor. In 17 patients, no change in CEA levels occurred in association with the clinical re-establishment of recurrent or metastatic tumor. The interval between the occurrence of levels of plasma CEA in excess of 40 ng/ml and the clinical detection of recurrence or metastasis was commonly around 9 months, although in one case this period was 18 months. The site of the metastases influenced whether raised plasma CEA values would develop or not. Most subjects with hepatic secondary tumors had raised levels. Raised levels were less likely with pelvic and peritoneal metastases and were highly unlikely in cases of local recurrence. The precise preoperative CEA level in plasma did not act as a clear prognostic index with regard either to the incidence of recurrences or the free interval before their detection. Dukes's staging remained a more accurate index. However, within any particular Dukes's stage, there was a tendency for the higher plasma values to carry a worse prognosis. Of greater importance was the postoperative failure of CEA levels to return to normal, which indicated spread outside the apparent pathological stage and a high and early rate of recurrence. Use of serial plasma CEA assays is recommended as an aid for the earlier detection of recurrent or metastatic colorectal carcinomas.

2099 INVASIVE AND METASTASIZING CARCINOMA IN A SMALL ADENOMATOUS POLYP OF THE COLON: REPORT OF A CASE WITH DEMONSTRATION OF A TUMOR ASSOCIATED ANTIGEN. *(E.)* Strauss, R. A.; Pascal, R. R. (Presbyt. Hosp., New York, N.Y.). *Hum. Pathol.* 6(2): 256-259, 1975.

An unusual case of metastasis of a carcinoma arising from an adenomatous polyp, without invasion of the pedicle, is presented. A 40 yr old woman presented with rectal bleeding. Sigmoidoscopy revealed a pedunculated lesion about 1 cm in diameter, 15 cm from the anal verge. A 0.3 cm portion of the head was removed, and examination showed a focus of moderately differentiated adenocarcinoma penetrating into the muscularis mucosae. A 30 cm segment of sigmoid colon was removed, on which a solitary polyp, 1.7 cm in diameter, on a 1.7 cm pedicle was found, 7 cm from the distal edge. No additional carcinoma was found in the polyp or intestinal wall; however, 2 lymph nodes adjacent to the wall were replaced with metastatic carcinoma. The carcinoma and atypical mucosal glands in the preliminary biopsy specimen, as well as the lymph node tumors, contained tumor associated antigen, while the remainder of the polyp and the surrounding bowel did not. The case refutes the concept that polyps giving rise to adenocarcinoma are polypoid carcinomas from the outset and products of transformation.

2100 FAECAL BILE-ACIDS AND CLOSTRIDIA IN PATIENTS WITH CANCER OF THE LARGE BOWEL. *(E.)* Hill, M. J.; Drasar, B. S.; Williams, R. E. O.; Meade, T. W.; Cox, A. G.; Simpson, J. E. P.; Morson, B. C. (Cent. Public Health Lab., London, England). *Lancet* 1(7906):535-538, 1975.

The fecal bile acid concentrations and presence or absence of nuclear dehydrogenating clostridia were compared in patients with and without large bowel cancer. Sixteen patients with colonic and 12 with rectal cancer were compared to 90 controls (50 with upper alimentary tract disease, 12 with non-malignant large bowel disease, and 28 with non-gastrointestinal disease). Eighty two percent of the cancer patients had fecal bile acid levels of 6 mg/g dry weight feces or over, compared with 17% of the control group. The same percentage of the cancer patients had nuclear dehydrogenating clostridia, compared with 43% of the controls. Seventy percent of the cancer patients had both the clostridia and the high fecal bile acid levels, compared with 9% of the controls. Differences were consistent for the different control subdivisions. Further study in 14 cancer patients and 41 controls showed a significantly higher proportion of mono and unsubstituted bile acids in the cancer patients, possibly indicating more extensive bacterial degradation. These findings support the hypothesis that high concentrations of bile acid derivatives produced by certain anaerobic bacteria play an important etiological role in large bowel cancer.

2101 CLINICAL EXPERIENCES AT ST. MARK'S HOSPITAL WITH MULTIPLE SYNCHRONOUS CANCERS OF THE COLON AND RECTUM. *(E.)* Heald, R. J.; Bussey, H. J. R. (St. Mark's Hosp., London, England). *Dis. Colon Rectum* 18(1):6-10, 1975.

Cases of double synchronous cancers of the large intestine at St. Mark's Hospital from 1928 to 1970 were analyzed. Synchronous growths occurred in 3.5% of cancer resections. The probability that a secondary malignancy would develop subsequent to resection of an initial malignant growth was also 3.5%. Eighty-two percent of the patients in this series had radical removal of both growths, 12% had palliative removal, and 6% were considered inoperable. The 5-yr survival rate after radical resection of double carcinoma was 66%. Following resection of 2 colorectal cancers, 4.3% of patients subsequently developed a third growth (average time 12.5 yr). The risk of developing metachronous growth rose from 3.5% after resection of a single cancer to more than 8% after removal of 2 growths. Cases of triple synchronous carcinoma demonstrated the same average survival time (7 yr 8 months) as that for single cancers. Survival rates based upon stage of malignancy development were: 82.8% after a single Dukes' A tumor; 88% after a double Dukes' A; 62.9% after a single Dukes' B; 66% after a double Dukes' B; 27.8% after a single Dukes' C; 25% after a double Dukes' C. A high incidence (75%) of malignancy was associated with benign tumors; in 27% of the cases, the malignancy was found within a pre-existing benign tumor. Since secondary growths are usually not palpable at operation, the need for detecting them by full clinical and radiologic investigations before any resection of the colon or rectum is emphasized.

2102 MUSCLE SPASM IN SIGMOID DIVERTICULOSIS: EVALUATION OF RETROGRADE COLON OBSTRUCTION BY HYPOTONIC BARIUM ENEMA. *(E.)* Ferrucci, Jr., J. T.; Jaffer, F.; Seidler, R. (Harvard Med. Sch., Boston, Mass.). *J. Can. Assoc. Radiol.* 25(4):269-274, 1974.

Eleven patients with complete retrograde sigmoid obstruction, visualized by routine barium enema, were given the anticholinergic drug propantheline bromide i.m. at 30 mg. Five min after injection a repeat enema was carried out. In 10 of the patients, an obstructing smooth muscle spasm was relaxed, and a preliminary diagnosis of diverticular disease was confirmed in each case. In the eleventh case the drug had no effect, and the resulting diagnosis of possible carcinoma was confirmed on surgery. It is concluded that this technique is useful in diagnosis of uncomplicated diverticular disease, and may avoid unnecessary surgery.

2103 MESENTERIC ARTERIAL INFUSIONS OF VASOPRESSIN FOR HEMORRHAGE FROM COLONIC DIVERTICULOSIS. *(E.)* Athanasoulis, C. A.; Baum, S.; Rosch, J.; Waltman, A. C.; Ring, E. J.; Smith, Jr., J. C.; Sugarbaker, E.; Wood, W. (Massachusetts Gen. Hosp., Boston). *Am. J. Surg.* 129(2):212-216, 1975.

Hemorrhage from colonic diverticulitis was reviewed in 24 cases with respect to recurrence in patients with angiographic therapy only, and in those with additional segmental resection. In each patient after extravasation was located by mesenteric arteriography, 0.2 U/min vasopressin was infused into the supplying artery for 6-12 hr, then 0.1 U/min for 12-24 hr. In 2 patients, the vasopressin was ineffective and surgery was required. One survived and had no recurrance after 10 months. Of the remaining 22, 5 underwent elective colonic resection, 5 had the surgery after recurrence within 6 days, and 12 underwent no surgery. The 5 who had elective surgery had no recurrences after follow-ups of 8-18 months. The 5 who underwent surgery for bleeding had no recurrences after follow-ups of 1-22 months. Of the 12 unoperated patients, 9 had no recurrences after 7-34 months. Two had recurrences at 2 and 4 months and were given surgery. The 3rd was brought under control with conventional drug therapy. It is concluded that mesenteric angiography effectively localizes the bleeding site and directly aids in vasopressin therapy. The resulting hemostasis creates optimal surgical conditions, and the segmental colectomy made feasible by this technique is as effective as subtotal colectomy.

2104 A DOUBLE-BLIND COMPARISON OF THE EFFECTIVENESS OF AZATHIOPRINE AND SULFASALAZINE IN IDIOPATHIC PROCTOCOLITIS: PRELIMINARY REPORT. *(E.)* Caprilli, R.; Carratu, R.; Babbini, M. (2nd Med. Clin., Univ. Rome, Italy). *Am. J. Dig. Dis.* 20(2): 115-120, 1975.

The effectiveness of azathioprine was compared with that of salicylazosulfapyridine (sulfasalazine) in 20 patients with the active phase of idiopathic proctocolitis. In the double blind study, patients were paired according to the severity of the disease, and one of each pair was treated with azathioprine (2.5 mg/kg) or salicylazosulfapyridine (65 mg/kg) daily for 3 months. Progress of the disease was checked by endoscopy, radiography, and biopsy. Both drugs caused similar, statistically significant improvement of clinical symptoms, and there was no significant difference in their effects upon endoscopic, histologic, or radiological data. Both endoscopic and histological findings showed significant improvement, but radiological findings were less marked. Hematological analysis showed a significant increase in serum protein after salicylazosulfapyridine treatment. The patients on sulfasalazine showed no side effects, but of those on azathioprine, 3 developed anorexia and 1, leukopenia. These were not serious enough to warrant discontinuation of treatment. It is concluded that azathioprine may be used in colitis when salicylazasulfapyridine is contraindicated, and that radiological findings do not necessarily correlate closely with clinical improvement.

2105 PSEUDOMEMBRANOUS ENTEROCOLITIS. RESURGENCE RELATED TO NEWER ANTIBIOTIC THERAPY. *(E.)* Keeffe, E. B.; Katon, R. M.; Chan, T. T.; Melnyk, C. S.; Benson, Jr., J. A. (Univ. Oregon Med. Sch., Portland). *West. J. Med.* 121(6):462-472, 1974.

Six patients with pseudomembranous enterocolitis were seen at one institution over a 6-month period. Clindamycin therapy preceded the diagnosis in all 6 cases and possibly caused the disease in 5. The patients, 5 men and 1 woman aged 35-75 yr, were admitted for the following reasons and received the antibiotic therapy indicated. A diabetic underwent amputation of the lower left leg due to persistent ulceration. He was given postoperative nafcillin (4 g i.v./day) and gentamycin (180 mg i.m./day) for 10 days, plus 2 doses of clindamycin (total 1,200 mg i.v.) on the second postoperative day. A woman received a 10-day course of ampicillin p.o. for gastroenteritis. Toxic megacolon developed and, beginning the day before subtotal colectomy, she received ampicillin (8 g i.v./day), clindamycin (2,400 mg i.v./day) and gentamycin (240 mg i.m./day). A case with chronic obstructive pulmonary disease and squamous cell carcinoma of the larynx received procaine penicillin (1,200,000 U i.m./day) for 210 days. A laryngectomy with radial neck dissection was performed. Starting the day of operation, he received clindamycin (1,800 mg/day for 7 days, 3 days i.v. and 4 p.o.). A man with peripheral vascular disease had a left-to-right femoral bypass graft. Postoperative antibiotics included cephalosporin (4 g i.v./day for 4 days), aqueous penicillin (6 million U/day i.v. for 8 days), gentamycin (240 mg i.m./day for 3 days), and clindamycin (2,400 mg/day i.v. for 6 days). A man with paraesophageal hiatal hernia and chronic atrial fibrillation secondary to mitral valve and coronary artery disease received clindamycin (total 1,200 mg i.v. in 2 doses), and kanamycin (1,000 mg i.m.) the day before operation and aqueous penicillin (6 million U/day i.v.) for 2 postoperative days. A man with pneumonia received clindamycin (2,400 mg/day i.v. for 6 days). Common clinical features of the pseudomembranous entercolitis which developed in

each patient included diarrhea, abdominal pain, fever, leukocytosis, radiographic findings of large bowel dilation with mucosal thickening and characteristic sigmoidoscopic or gross pathologic demonstration of discrete yellow-white plaques on otherwise normal mucosa. Complications included toxic megacolon and sigmoid colon perforation. Of the 6, 2 died. By virtue of these cases and a literature review, lincomycin and clindamycin are implicated in the recent resurgence of this formerly rare variety of colitis.

2106 ANORECTAL ANOMALIES. *(E.)* Nixon, H. H. (Hosp. Sick Child., London, England). *Leban. Med. J.* 27(4):407-411, 1974.

2107 ACTIONS OF BRAN ON COLONIC MOTILITY RELATED TO ITS PHYSICAL PROPERTIES. *(E.)* Kirwan, W. O.; Smith, A. N.; McConnell, A. A.; Mitchell, W. D.; Eastwood, M. A. (Dep. Clin. Surg., Univ. Edinburgh, Scotland). *Gut* 15(10):828, 1974.

2108 OPERATIVE COLOSCOPY: A CURRENT STATUS APPRAISAL. *(E.)* Marks, G. (Med. Coll., Thomas Jefferson Univ., Philadelphia, Pa.). *Dis. Colon Rectum* 17(6):754-758, 1974.

2109 FREE PERFORATION OF THE COLON. ETIOLOGY AND TREATMENT IN 44 CASES. *(E.)* Huttunen, R.; Larmi, T. K. I.; Heikkinen, E.; Rasanen, O. (Oulu Univ. Cent. Hosp., Finland). *Acta Chir. Scand.* 140(7):535-541, 1974.

2110 SPONTANEOUS PERFORATION OF THE COLON. *(Ger.)* Seitz, W.; Forster, C.; Rothmund, M. (Surg. Clin., Univ. Mainz, Germany). *Therapiewoche* 24(18):2043-2048, 1974.

2111 CHOLECYSTOCOLIC FISTULA WITH COLONIC OBSTRUCTION: REPORT OF A CASE. *(E.)* Balsano, N. A.; Reynolds, B. M. (Misericordia Hosp. Med. Cent., Bronx, N.Y.). *Dis. Colon Rectum* 17(6): 766-768, 1974.

2112 PSEUDO-OBSTRUCTION OF THE LARGE BOWEL. *(E.)* Bardsley, D. (R. Hosp., Sheffield, England). *Br. J. Surg.* 61(12):963-969, 1974.

2113 ACUTE DILATATION OF THE LARGE INTESTINE IN PATIENTS WITH PROCTOCOLITIS. *(Ger.)* Novak, J.; Nedbal, J.; Maratka, Z.; Hledik, E. (Inst. Postgrad. Med. Pharm., Prague, Czechoslovakia). *Dtsch. Z. Verdau. Stoffwechselkr.* 34(1):45-51, 1974.

2114 TREATMENT OF CHRONIC PROCTITIS WITH DISODIUM CROMOGLYCATE. *(E.)* Heatley, R. V.; Calcraft, B. J.; Rhodes, J.; Owen, E.; Evans, B. (Univ. Hosp. Wales, Cardiff). *Gut* 15(10):829, 1974.

2115 IRRITABLE COLON. *(Ger.)* Nord, H. J. (Dept. Internal Med., Univ. S. Florida, Tampa). *Therapiewoche* 24(22):2565-2569, 1974.

2116 MEGACOLON IN ADULTS: LITERATURE REVIEW. *(Rus.)* Fedorov, V. D.; Gelfenbein, L. S.; Meituv, M. B. (Sci. Res. Lab. Proctol., Moscow, USSR). *Khirurgiia (Mosk.)* (11):136-140, 1973.

2117 IDIOPATHIC COLONIC ULCER: REPORT OF A CASE DIAGNOSED PREOPERATIVELY. *(E.)* Eusebio, E. B.; Kaplan, A. P. (Mt. Sinai Hosp., Minneapolis, Minn.). *Dis. Colon Rectum* 17(6):763-765, 1974.

2118 THE PRESENT STATUS IN HIRSCHSPRUNG'S DISEASE. *(E.)* Nixon, H. H. (Hosp. Sick Child., London, England). *Leban. Med. J.* 27(4):401-405, 1974.

2119 TWO CASES OF COLONIC FAMILIAL MULTIPLE POLYPOSIS IN CHILDREN. *(E.)* Ortiz, J.; Guastavino, E.; Toccalino, H. (Professor A. Posadas Polyclin., Haedo, Argentina). *Acta Gastroenterol. Latinoam.* 6(1):47-50, 1974.

2120 CHRONIC CONSTIPATION AND ULTRA-SHORT SEGMENT HIRSCHPRUNG'S DISEASE IN CHILDREN: DIAGNOSIS AND MANAGEMENT. *(E.)* Shandling, B. (Hosp. Sick Child., Toronto, Canada). *Paediatrician* 3(3/5): 213-219, 1974.

2121 ROENTGENOGRAPHIC FINDINGS OF COLO-RECTAL CARCINOMA AND ITS PROGNOSIS. *(E.)* Satoh, K. (Tohoku Univ. Sch. Med., Sendai, Japan). *Asian Med. J.* 17(2):65, 1974.

2122 CANCER OF THE CECUM: A REAPPRAISAL. *(E.)* Adam, Y. G.; Stern, H.; Volk, H. (Albert Einstein Med. Coll., Yeshiva Univ., Bronx, N.Y.). *Isr. J. Med. Sci.* 10(12):1536-1540, 1974.

2123 STATISTICAL AND ETIOLOGICAL ASPECTS OF CARCINOMA OF THE LARGE INTESTINE. A STUDY BASED ON AUTOPSY DATA FROM 1928-1972. *(E.)* Parkash, O. (Wilhelminen Hosp., Vienna, Austria). *Digestion* 11(1/2):115-127, 1974.

2124 MALIGNANCIES OF THE LARGE INTESTINE IN PERSONS LESS THAN 20 YEARS OLD: REPORT OF TWO CASES. *(E.)* Goldman, H. S.; Smith, D. L.; Foote, R. F. (Foote-Smith Proctol. Med. Group Inc., Encino, Calif.). *Dis. Colon Rectum* 17(6):771-776, 1974.

2125 CARCINOMA OF THE COLON ASSOCIATED WITH TUBERCULOSIS: REPORT OF A CASE. *(E.)* Tandon, H. D.; Kapoor, B. M. L. (All-India Inst. Med. Sci., New Delhi). *Dis. Colon Rectum* 17(6):777-781, 1974.

2126 CAVERNOUS HEMANGIOMA OF THE RECTUM AND APPENDIX: REPORT OF A CASE. *(E.)* Harned, R. K.; Dobry, C. A.; Farley, G. E. (Univ. Nebraska Sch. Med., Omaha). *Dis. Colon Rectum* 17(6):759-762, 1974.

2127 GASTROINTESTINAL HEMANGIOMATOSIS. *(Rus.)* Krysko, V. S.; Shavarov, I. G. (Volynsk Reg. Hosp., Lutsk. USSR). *Klin. Khir.* (10):45, 1974.

2128 INTEGRATION OF CHEMOTHERAPY INTO COMBINED MODALITY TREATMENT OF SOLID TUMORS. II. LARGE BOWEL CARCINOMA. *(E.)* Carter, S. K. (Natl. Cancer Inst., Bethesda, Md.) *Cancer Treat. Rev.* 1(2):111-129, 1974.

2129 A COMPARISON OF RESTORATIVE RESECTION OF CARCINOMA OF THE MIDDLE THIRD OF THE RECTUM WITH ABDOMINOPERINEAL EXCISION. *(E.)* Penfold, J. C. B. (Dep. Surg., Univ. Melbourne, Australia). *Aust. NZ J. Surg.* 44(4):354-356, 1974.

2130 DUSTING OF THE MUCOSA IN THE DISTAL LARGE INTESTINE WITH DRY DRUGS. *(Rus.)* Shatyrko, A. S.; Zhukov, A. V. (Truskavets Sanitorium, USSR). *Vrach. Delo* (11):90-92, 1974.

See also, 1885, 1899, 1901, 1902, 1903, 2045, 2046, 2047, 2048, 2055, 2059, 2080, 2133, 2339, 2381, 2397.

LARGE INTESTINE

Ulcerative Colitis

2131 HISTOLOGICAL LIVER DISORDER AND ANTI-COLON ANTIBODIES IN ULCERATIVE COLITIS. *(E.)* Marcussen, H. (Gentofte Hosp., Hellerup, Denmark). *Acta Pathol. Microbiol. Scand.* [A] 82(6):714-718, 1974.

The possibility of a correlation between the presence of circulating anti-colon antibodies determined by the indirect immunofluorescent technique and the occurrence of abnormal liver histology in ulcerative colitis was investigated in 97 patients (56 women and 41 men, aged 16-74 yr). Evidence of histological liver changes was present in 39 patients (40%) and included mild degrees of inflammation (14), periportal inflammation (20), chronic pericholangitis (3), cirrhosis (1), focal necrosis (1), and fatty change (7). Anti-colon circulating antibodies were found in 9 patients (19%), in 7 of whom abnormal liver histology was found including mild cell infiltration in 1 case, periportal inflammation in 5 and cirrhosis in 1. Using the described histological parameters of liver disorder, no correlation with the presence of anti-colon antibodies was seen. Varying degrees of cholangitis were noted in 23 cases, but only 5 of these patients (22%) had antibodies in the blood, i.e., the same occurrence in the cholangitis group as in the total patient group. That this study failed to demonstrate a correlation between liver disorder and circulating anti-colon antibodies speaks against the "malsequestrating" theory. It is suggested that factors other than severity of disease influence the ability to develop antibodies.

2132 COAGULATION STUDIES IN ULCERATIVE COLITIS AND CROHN'S DISEASE. *(E.)* Lam, A.; Borda, I. T.; Inwood, M. J.; Thomson, S. (St. Joseph's Hosp., London, Canada). *Gastroenterology* 68(2):245-251, 1975.

The hemostatic mechanisms of 12 patients (5 men and 7 women, aged 21-76 yr) with ulcerative colitis and 12 (4 men and 8 women, aged 17-52 yr) with Crohn's disease were compared to 19 patients with other diseases and to 15 healthy controls. The tests in which no significant differences were found included: hemoglobin, hematocrit, bleeding time, WBC, prothrombin time, partial thromboplastin time, thrombin time, fibrinogen degradation products, protamine sulfate, thrombin generation, and euglobulin lysis time. Test results showing significant differences for ulcerative colitis, Crohn's disease, hospitalized controls and normal controls were: platelets (x 10^3), which were 341, 282, 212, and 194, resp.; factor VIII (%) which were 92.3, 212, 94, and 88, resp.; factor V (%) which were 129, 131, 102, and 94, resp.; fibrinogen (mg/100 ml) which were 427, 433, 317, and 300, resp.; and antithrombin III (U) which were 62.1, 68, 88, and 78.5, resp. These findings suggest that patients with ulcerative colitis, as well as patients with Crohn's disease, may well be predisposed to thromboembolic events because of changes in the hemostatic mechanism. The data indicate that the low antithrombin III level may actually reflect excessive interaction with thrombin, rather than diminution in production of this protein by a damaged liver. The essentially normal bleeding times for the patients indicate that the platelets were functioning normally. It is concluded that the thrombocytosis in these patients was related to bowel disease. It is suggested that individuals with chronic inflammatory diseases of the bowel may produce larger amounts of coagulation factors with a potential increase in thrombin production. Thus, it is suggested that patients with the above mentioned changes be considered as a high risk group and be managed accordingly.

2133 COLONOSCOPY IN INFLAMMATORY BOWEL DISEASE. *(Fr.)* Crespon, B.; Housset, P.; Debray, C. (Bichat Hosp., Paris, France). *Ann. Gastroenterol. Hepatol. (Paris)* 9(6):529-535, 1973.

Colonoscopic examinations were performed on 26 patients with ulcerative colitis; 12 with Crohn's disease of the colon; 4 with poorly defined ulcerative colitis, including 2 with malignant tumors located below the inflammation; 1 with amebic colitis; and 3 with radiation-induced colitis. Ulcerative colitis was characterized by congestion, mucosal fragility, bleeding, and continuous lesions without areas of normal mucosa. In advanced forms the colonic mucosa had been replaced by multiple pseudopolyps, the tops of which coalesced. In Crohn's disease of the colon, the lesions were located in the sigmoid and were segmental in distribution. Aphthoid ulcers were found in some mild cases, while small inflammatory polyps developed around ulcerations in chronic cases. In contrast to pseudopolyps in ulcerative colitis, these pseudopolyps were less numerous, not as highly developed, and did not coalesce at the top. However, to establish a differential diagnosis between ulcerative colitis and Crohn's disease, it is necessary to take into account clinical, radiological, endoscopic, and biopsy findings. Radiation-induced colitis was characterized by a mucosa which was pale, sclerotic, telangectatic, difficult to biopsy, and which bled easily. A case of subacute amebic colitis was characterized only by the nonhemorrhagic nature of the lesions; the diagnosis was established by parasitological examination of the stools and mucus taken during endoscopic examination. In 2 patients with ulcerative colitis located above neoplasms of the left colon, multiple, superficial, nonhemorrhagic ulcerations disappeared several months after removal of the tumors. These lesions might have been caused either by stasis or ischemia. Colonoscopy enabled a diagnosis of Behcet's disease to be made in a patient with ulcerative lesions in the left colon which were not visualized radiologically. Followup colonoscopic examinations showed complete disappearance of ulcerations in 2 patients with ulcerative colitis and, 3 months after corticosteroid therapy, in 2 patients with Crohn's disease of the colon.

2134 EFFECTS OF SULPHASALAZINE (SALAZOPYRIN) ON FAECAL FLORA IN PATIENTS WITH INFLAMMATORY BOWEL DISEASE. *(E.)* West, B.; Lendrum, R.; Hill, M. J.; Walker, G. (St. Mary's Hosp., London, England). *Gut* 15(12):960-965, 1974.

The effects of salicylazosulfapyridine on the fecal flora of 26 patients with inflammatory bowel disease was investigated. Twenty one patients had proctocolitis, of whom 6 had acute exacerbation and 3, complete clinical remission. Prior to the study 11 had been taking salicylazosulfapyridine intermittently for periods of 2 months to 14 yr at 1-3 g daily. Five patients with Crohn's colitis were also studied, one of whom was treated with the drug. No significant differences were found between bacterial counts from 4 patients with Crohn's colitis and 6 with proctocolitis, none of whom had received the drug. Proctocolitis patients with acute exacerbation showed greater numbers of all microbes than those with less active disease, but not to a significant degree. Comparison of the 10 patients who had either condition and had not received the drug with the 5 who had received it throughout showed a significant decrease in the aerobic bacterial count of the latter largely due to reduction of the enterobacteria (7.8 $\log_{10}$ mean bacterial counts/ml feces with no drug; 6.6 with drug). Total anaerobes were reduced from counts of 9.9 to 9.1. In patients who received the drug at some time, but not continually, significant reductions were also found in the enterobacteria (40 fold), in total non-sporing anaerobes (20 fold), and in opalescent-negative clostridia (200 fold). It is concluded that salicylazosulfapyridine causes a consistent decrease in both aerobic and anaerobic bacteria.

2135 SERUM COMPLEMENT COMPONENTS C_3 AND C_4 IN INFLAMMATORY BOWEL DISEASE. *(E.)* Ward, M.; Eastwood, M. A. (West. Gen. Hosp., Edinburgh, Scotland). *Gut* 15(10):835, 1974.

2136 CEA ESTIMATION IN ULCERATIVE COLITIS WITH AND WITHOUT MALIGNANT CHANGE. *(E.)* Dilawari, J. B.; Lennard-Jones, J. E.; Mackay, A. M.; Ritchie, J. K.; Sturzaker, H. G. (St. Mark's Hosp., London, England). *Gut* 15(10):828, 1974.

2137 THE EXTENT OF RECTAL PREMALIGNANT CHANGE IN ULCERATIVE COLITIS. *(E.)* Riddell, R. H. (St. Mark's Hosp., London, England) *Gut* 15(10): 822, 1974.

2138 CONSERVATIVE TREATMENT OF ULCERATIVE COLITIS AS SEEN BY PRECISE OBSERVATIONS. *(Pol.)* Kociecka, W.; Goral, R. (Acad. Med., Poznan, Poland). *Przegl. Lek.* 31(3):408-413, 1974.

2139 IMMUNOSUPPRESSIVE CONSEQUENCES OF THE TREATMENT OF ULCERATIVE COLITIS WITH AZATHIOPRINE. *(E.)* Skinner, J. M.; Campbell, A. C.; Waller, C.; Wood, J.; Maclennan, I. C. N. (Radcliff Infirm., Oxford, England). *Gut* 15(10):828-829, 1974.

2140 AMYLOIDOSIS AND GRANULOMATOUS ILEOCOLITIS. REGRESSION AFTER SURGICAL REMOVAL OF THE INVOLVED BOWEL. *(E.)* Fitchen, J. H. (U.S. Air Force Clin., Norton Air Force Base, Calif.). *N. Engl. J. Med.* 292(7):352-353, 1975.

2141 ILEOANOSTOMY IN ULCERATIVE COLITIS (RAVITCH PROCEDURE). *(E.)* Fujiwara, Y.; Ohizumi, T.; Kakizaki, G.; Fujiwara, T. (no affil.). *J. Pediatr. Surg.* 10(1):145-146, 1975.

2142 PSYCHOLOGICAL FACTORS IN A DISEASE SIMILAR TO ULCERATIVE COLITIS IN A KERRY BLUE AND A SKYE TERRIER. *(Ger.)* Adler, R.; Troup, S.; Engel, G. L. (Med. Clin., Univ. Bern, Switzerland). *Z. Gastroenterol.* 12(3):191-192, 1974.

2143 JUSTIFICATION OF DISPENSARY THERAPY FOR CHRONIC INTESTINAL DISEASES. *(Ger.)* Kettner, W. (Bitterfeld Reg. Hosp., Germany). *Z. Aerztl. Fortbild.* 68(6):305-307, 1974.

See also, 2114, 2388, 2395, 2398.

PANCREAS

2144 THE FATE OF HETEROTOPIC PANCREATIC TISSUE. A STUDY OF 212 CASES. *(E.)* Dolan, R. V.; ReMine, W. H.; Dockerty, M. B. (Mayo Clin., Rochester, Minn.). *Arch. Surg.* 109(6):762-765, 1974.

A survey was made of all records of patients in whom the diagnosis of heterotopic pancreas had been made over a 20 yr period in order to determine whether the mass of aberrant pancreatic tissue is, by itself, necessarily a serious matter. A total of 212 patients (136 males and 76 females) were so diagnosed. The largest incidence of discovery occurred in the 6th decade of life. The tissue was found in the stomach (81 cases), duodenum (77), upper part of the jejunum (33), Meckel's diverticulum (11), ileum (3), and 1 each in the common bile duct, umbilicus, gallbladder, hilus of spleen, regional papilla of accessory pancreatic duct, near left gastric artery, and anterior to pancreas. In all but 33 patients, the tissue was found at operation, usually for other conditions. These other 33 patients were diagnosed roentgenographically. A firm causal relation to symptoms was difficult to establish. Obscure gastrointestinal tract bleeding, often attributed to this lesion, was rarely associated with ulceration or necrosis when the lesion was excised and examined microscopically. When heterotopic pancreas was discovered at operation, however, excision was a safe and warranted procedure. It was concluded that heterotopic pancreatic tissue was not responsible for the initial features in the majority of these cases. It is noted that remission of symptoms can be expected as frequently with expectant observation as with surgical intervention. The only obvious exception to this would be patients such as the 3 in this study who had suspected gastrointestinal tract blood loss and who had no such difficulties after operation.

2145 SUPRADIAPHRAGMATIC ASPECTS OF BENIGN PANCREATIC DISEASE. *(E.)* Walton, III, T. P.; Quinilty, R. J. (Louisiana State Univ. Sch. Med., New Orleans). *Am. Surg.* 41(1):32-36, 1975.

A classification of supradiaphragmatic extension of benign pancreatic disease is proposed, based on 74 cases from the literature and 3 new cases (2 men and 1 woman, aged 29-59 yr). The 3 main categories include: pleural accumulation of amylase-rich fluids; direct extension of the pseudocyst; and fistula formation. The category of pleural accumulation of amylase-rich fluids is further subdivided into pancreatothorax, including cases associated with acute pancreatitis, chronic pancreatitis or pseudocyst; pancreatohemothorax; and pancreatochylothorax. The category dealing with the direct extension of the pseudocyst includes mediastinal pseudocyst and cervical pseudocyst. The final category, fistula formation, considers pancreatobronchial fistula and pancreatopleural fistula, the latter being divided into direct and indirect.

2146 EXPERIMENTAL ALLOTRANSPLANTATION OF THE PANCREAS IN DOGS: HISTOPATHOLOGICAL CHANGES. *(Por.)* Ellenbogen, G.; Raia, A. A.; Behmer, O. A.; Pollara, W. M.; Zilberstein, B.; Morimoto, R. Y.; Neto, O. B.; Pinotti, H. W. (Fac. Med., Univ. Sao Paulo, Brazil). *Rev. Hosp. Clin. Fac. Med. Sao Paulo* 29(3):124-132, 1974.

Rejection was studied in 10 male mongrel dogs who underwent total pancreatectomy 5 days before receiving an allotransplant of duodenum and pancreas. The celiac trunk of the explant was anastomosed end-to-end to the proximal segment of the primary iliac artery. Duodenal continuity was reestablished by an end-to-side anastomosis of the distal duodenum of the explant to the lower part of the mid-duodenum in the recipient. Recipients were not given immunosuppressive agents but did receive oxytetracycline (15 mg/kg/day i.m.) postoperatively. Serial biopsies of the transplanted pancreas, taken at 48 hr intervals, revealed intramural fibrin deposits in pancreatic arterioles 4 days after surgery; these were followed by localized fibrinoid arteriolar necrosis on days 6-8. Starting on the 6th day, infiltrates of lymphocytes and plasma cells appeared, mostly around the blood vessels, together with interstitial connective tissue. While no appreciable lesions were observed in the endocrine cells, diffuse necrosis suddenly developed in the exocrine glands at the time of rejection.

2147 AN ACCESSORY PANCREAS IN THE WALL OF THE DUODENUM. *(Rus.)* Chuprina, V. V.; Zaporozhets, V. K. (Voroshilovgrad Med. Inst., USSR). *Khirurgiia (Mosk.)* (3):114-115, 1974.

2148 PANCREATIC DISEASE IN CHILDREN. *(Por.)* Mott, C. de B. (Clin. Hosp., Sao Paulo, Brazil). *Arq. Gastroenterol. Sao Paulo* 11(4):229-235, 1974.

2149 TREATMENT OF CANCER OF THE EXOCRINE PANCREAS. *(Fr.)* Clot, J. P.; Bacourt, F.; Mercadier, M. (Hop. de La Pitie, Paris, France). *Cah. Med.* 15(2):107-110, 1974.

2150 STUDIES ON THE EXTERNAL PANCREATIC SECRETION. *(E.)* Henriksen, F. W. (Inst. Exp. Res. Surg., Univ. Copenhagen, Denmark). *Scand. J. Gastroenterol.* [*Suppl.*] 9:1974.

2151 BIOCHEMICAL STUDIES OF PURE PANCREATIC JUICE OBTAINED BY DUODENOSCOPIC CANNULATION OF THE PANCREATIC DUCT IN CONSCIOUS PATIENTS. *(E.)* Cotton, P. B.; Cremer, M.; Robberecht, P.; Christophe, J. (St. Thomas's Hosp., London, England). *Gut* 15(10): 838, 1974.

See also, 1756, 1886, 1887, 1900, 1904, 1905, 1906, 1909, 2054, 2334, 2339, 2341.

PANCREAS

Pancreatitis

2152 GALLBLADDER AND PANCREAS. *(Ger.)* Neumayr, A.; Peschl, L. (Kaiserin Elisabeth Hosp., Vienna, Austria). *Leber Magen Darm* 3(1):23-28, 1973.

The anatomy and physiology of the pancreas and gallbladder are reviewed and pathoanatomical and pathophysiological aspects of pancreatitis are presented. The draining ducts of the gallbladder and pancreas have anatomically and functionally close relations, with the common bile duct leading to the duodenum in 70-90% of all cases. Under normal conditions, the pressure in the secretion and rest phases is higher in the pancreatic duct than in the choledochus, and consequently the pressure increase occurring in pancreatic duct obstruction makes a pancreatic bile reflux possible. The frequent coincidence of acute pancreatitis with bile duct diseases is due to the activation of proteolytic enzymes and deconjugation of bile salts by bile infected with *E. coli*, clostridia, and enterococci. Trypsin, elastase, and phospholipase A are known to play an important role in the genesis of acute pancreatitis. Vasoactive trypsin impairs the microcirculation in the pancreatic tissue and may lead to serious hemorrhagic pancreatitis. Obstruction of the draining ducts is revealed in about 50% of all cases of chronic pancreatitis. Pancreatitis may result also from cholecystitis conveyed through the lymph system. Secondary stenosis as a result of choledocholithiasis, shrinkage in the region of the sphincter of Oddi, or primary stenosis of unknown etiology, diffuse fibrosis of the pancreas with marked dilatation of the ducts, and susceptibility to episodes of acute pancreatitis are observed in chronic retention pancreatitis. Pain, exocrine pancreas insufficiency, steatorrhea, and loss of weight are the principal symptoms of chronic pancreatitis. Carcinoma of the head of the pancreas or of the papilla of Vater may cause obstruction of the bile duct. Acute cholecystitis is observed in 10% of all patients with acute pancreatitis. Chronic pancreatitis may be complicated by tubular stenosis of the retropancreatic segment of the choledochus, a condition that is very infrequently accompanied by obstructive jaundice.

2153 ELECTROCARDIOGRAPHIC CHANGES IN ACUTE PANCREATITIS. *(It.)* Zacco, R.; Aletti, P.; Martinotti, A.; Sala, G. (Polyclin. Hosp. Milan, Italy). *Atti Acad. Med. Lomb.* 27(1/2/3/4):87-119, 1972.

Electrocardiographic changes were found in 68 of 106 patients (58 men and 48 women) with acute hemorrhagic necrotizing, edematous, or cystic pancreatitis. All patients were in a state of shock; none had gallbladder or heart disease, fevers, or any other diseases which might have interfered with the findings. ECG changes were detected in 18 of 24 men (75%) less than 40 yr old, but in only 16 of 34 men (47%) over 40 yr old. The frequency of ECG changes was about the same in women over 45 yr (21 of 30 or 70%) and in those under 45 yr old (13 of 18 or 72%). The most common types of changes were sinus tachycardia (36 cases), localized ischemia (26 cases), and diffuse ischemia (22 cases). Atrial fibrillation occurred in 2 cases, extrasystole in 3, flutter in 1, and changes suggestive of myocardial infarction in 1 (a 27-yr-old woman). Of the 68 abnormal ECGs, 54 returned to normal within 1-15 days while 6 remained somewhat abnormal although the initial abnormalities did regress; 5 patients were lost to follow-up. From a review of the literature, it is concluded that ECG changes occurring in acute pancreatitis originate in the autonomic nervous system.

2154 THE SERUM COMPLEMENT SYSTEM -- A MEDIATOR OF ACUTE PANCREATITIS. *(E.)* Seelig, R.; Ehemann, V.; Tschahargane, C.; Seelig, H. P. (Inst. Pathol., Univ. Heidelberg, Germany). *Virchows Arch. [Pathol. Anat.]* 365(3):193-199, 1975.

The role of the complement system in the initial membrane lesion of acute pancreatitis was investigated. Acute pancreatitis was induced in 14 male Wistar rats by intraductal injection of 1 ml sodium desoxycholate. Ten minutes after injection a decline of about 20% of serum complement activity was noted. After 60 min the rate of decline reached about 35%; after 180 min, 60%. Analyzing single values allowed the differentiation of 2 groups of rats in this series. In the first group (6 rats), a decrease of serum complement activity to 26% of preoperative values was noted after 180 min. Five of these 6 rats did not survive a short narcosis for blood letting. The only surviving rat showed a 100% decline of complement activity after 360 min. In the second group (8 animals), the rate of decrease did not fall below 50% of the preoperative values. After reaching a minimum level within 3 hr, the serum complement activity rose again to 77% of the preoperative values in 6 hr. Histological examinations revealed that parenchymal damage of pancreatic tissue was related to the rate of decrease of serum complement activity. Progressive leukocytosis and acinar cell necrosis were observed when the rate of decline of complement components

was greater than 50% in 3 hr. The deposition of C3 component of complement in acute pancreatitis was demonstrated by immunofluorescence. To rule out mere deposition or activation of complement in the interstitial exudative fluid, single acinar cells of rat and rabbit pancreatic tissue were prepared and transferred to culture medium. In contrast to heat inactivated serum and C6 deficient serum, these cells were lysed by trypsin activated fresh serum. Consequently, an acute pancreatitis could be induced by activating exclusively the complement system by injection of cobra venom factor into the pancreatic duct of rats. The activated complement system may be responsible for initial membrane lesion in exudative inflammation, as could be shown in acute pancreatitis.

2155 PERITONEOCENTESIS AND PERITONEAL PERFUSION IN ACUTE PANCREATITIS: VALUE AND LIMITATIONS. *(Fr.)* Doutre, L. P. (no affil.). *Acta Chir. Belg.* (5):380-386, 1973.

Trypsin and lipase activities were determined in peritoneal fluid from 34 patients with acute abdomen who, at surgery, were found to have either peritonitis, edematous pancreatitis, or hemorrhagic necrotizing pancreatitis. The control group consisted of 16 patients with clinical symptoms of chronic pancreatitis. No significant differences were found in the trypsin activities, but lipase activities were significantly increased only in peritoneal fluid from patients with acute hemorrhagic necrotizing pancreatitis. Preliminary results suggest that if lipase activities in peritoneal fluid exceed 10 Vogel-Zieve U, the patient has pancreatic necrosis. In one patient who developed a fistula after left pancreatectomy for necrosis, both lipase and trypsin activities were still elevated the day before the fistula closed. In another patient, who underwent resection of the duodenum and head of the pancreas, activities of both enzymes decreased to zero over a 5-day period. It is recommended that determination of lipase activities in peritoneal fluid be performed on patients with acute abdomen to determine whether they have pancreatic necrosis and need immediate surgery.

2156 INTERACTIONS BETWEEN PANCREATITIS AND HYPERLIPOPROTEINEMIA. *(Ger.)* Dabels, J. (Med. Clin., Univ. Rostock, Germany). *Dtsch. Z. Verdau. Stoffwechselkr.* 33(4/5):213-221, 1973.

Attacks of acute pancreatitis and crises of abdominal pain were studied in 127 patients with essential hyperlipoproteinemia. Pancreatitis was found to be a complication of primary essential hyperlipoproteinemia in 8 cases. All patients belonged to types 4 and 5 according to Fredrickson. Disturbances in the fat metabolism in acute and chronic pancreatitis were investigated in 111 patients by the determination of the principal lipid groups and plasma proteins. The incidences of above-normal total lipid, triglyceride, cholesterol, and phospholipid levels were found to be 64%, 42%, 17%, and 11%, resp. The β-lipoprotein and pre-β-lipoprotein levels were 69.4% and 17.9% in acute, and 45.9% and 40.7% in chronic pancreatitis, resp. While the problem of the etiopathogenetic connection between pancreatitis and hyperlipoproteinemia is yet to be solved, the available data indicate that hyperlipoproteinemia may induce, or be a concomitant symptom of, pancreatitis.

2157 ACUTE HEMORRHAGIC PANCREATITIS IN THE DOG. 3. THE EFFECT OF ANTIDIURETIC HORMONE ON PANCREATIC TISSUE AND BODY FLUIDS. *(E.)* Schapiro, H.; Britt, L. G.; Blackwell, C. F.; Gaines, K. (Med. Unit, Univ. Tennessee, Memphis). *Am. Surg.* 41(2): 111-116, 1975.

The effects of antidiuretic hormone, Pitressin (0.25 U/kg/hr, i.v.) on pancreatic tissue and body fluids of 11 dogs subjected to closed duodenal loop obstruction for 24 hr were studied. A closed duodenal loop 10 cm long was created by occluding the duodenum with O-silk at the pylorus and a point distal to the ventral pancreatic duct. Blood supply to the pancreas and duodenal loop was preserved. Antidiuretic hormone modified the formation of acute hemorrhagic pancreatitis induced by the closed duodenal loop and changed the appearance of the duodenal loop. In untreated dogs, the closed duodenal loop resulted in pancreatic edema and acinar tissue disruption after 12 hr. In dogs treated with antidiuretic hormone, only a slight edema of the pancreas was noted after 24 hr. The closed loop elevated volume and amylase concentration in the peritoneal cavity. However, these increases were considerably less in the antidiuretic hormone treated animals. In untreated animals, the closed-loop fluid was viscous and foul-smelling with a high amylase concentration, and microscopic examination of the duodenal loop tissue revealed blood vessel engorgement, RBC extravasation and villi damage. The treated dogs had a lesser amount of fluid with a slight odor and an amylase concentration considerably less than untreated animals. Microscopically, the antidiuretic hormone-treated duodenal loop revealed blood vessel engorgement with some RBC extravasation, but little or no damage to the villi. In the untreated dogs, the closed loop caused elevated serum amylase after 18 hr and elevated serum lipase after 24 hr. Injections of antidiuretic hormone, i.v., did not modify either the time nor the extent of these enzyme increases, although the extent of pancreatic damage was significantly reduced. Thus, in these experiments, antidiuretic hormone reduced the intensity of the experimentally induced pancreatitis.

2158 CHRONIC PANCREATITIS: ETIOLOGICAL ASPECTS. OBSERVATION OF 50 CASES. *(Por.)* Mott, C. de B.; Ohki, Y.; Barros, M. I. B.; Conte, V. P.; Machado, M. C. C.; Bove, P.; Bettarello, A. (Fac. Med., Univ. Sao Paulo, Brazil). *Rev. Hosp. Clin. Fac. Med. Sao Paulo* 29(6):322-326, 1974.

2159 REGIONAL INCIDENCE OF CHRONIC AND RECURRENT PANCREATITIS. *(Ger.)* Herfort, K.; Bartos, V.; Kotrlik, J. (2nd Res. Inst. Gastroenterol., Charles Univ., Prague, Czechoslovakia). *Dtsch. Z. Verdau. Stoffwechselkr.* 33(6):293-297, 1973.

2160 CURRENT SURGICAL MANAGEMENT OF CHRONIC PANCREATITIS. *(E.)* Gaines, R. D. (Creighton Univ. Sch. Med., Omaha, Nebr.). *J. Natl. Med. Assoc.* 67(1):50-54, 1975.

2161 THE PATHOLOGY OF ENDOCRINE AND EXOCRINE PANCREATIC SECRETION IN PATIENTS WITH CHRONIC PANCREATITIS: INVESTIGATIONS WITH SECRETIN AND PANCREOZYMIN. *(Rus.)* Geller, L. I.; Kazimirchuk, A. P.; Romanets, V. A.; Bulgakova, O. S. (Khabarovsk Med. Inst., USSR). *Klin. Med. (Mosk.)* 52(7):40-45, 1974.

2162 ACUTE PANCREATITIS: PATHOLOGICAL, CLINICAL, AND BIOLOGICAL COMPARISONS. DIAGNOSTIC, PROGNOSTIC, AND THERAPEUTIC DEDUCTIONS (30 CASES). *(Fr.)* Barbas, A. (no affil.). *Can. Med.* 15(5):237-243, 1974.

2163 ACUTE PANCREATITIS ACCOMPANIED BY SHOCK AND KIDNEY FAILURE. *(Ger.)* Pichler, M.; Kleinberger, G.; Kotzaurek, R.; Pall, H.; Raberger, G.; Szeless, S. (1st Med. Clin., Univ. Vienna, Austria). *Wien. Klin. Wochenschr.* 87(3):83-87, 1975.

2164 THE RESULTS OF SOME LIVER FUNCTION TESTS IN PATIENTS WITH ACUTE PANCREATITIS. *(Rus.)* Suvalskaia, L. A.; Filin, V. I.; Kovalchuk, V. I. (I. I. Dzhanelidze Res. Inst. Emerg. Med. Treat., Leningrad, USSR). *Klin. Med. (Mosk.)* 52(11):67-70, 1974.

2165 CHANGES IN SERUM PROTEINS AND FREE AMINO ACID CONCENTRATIONS IN PERIPHERAL BLOOD VESSELS AND THE PORTAL SYSTEM IN PATIENTS WITH ACUTE PANCREATITIS AND CHOLECYSTOPANCREATITIS. *(Rus.)* Kovalev, M. M.; Tanasienko, I. D.; Lysenko, B. F. (A. A. Bohomolets Med. Inst., Kiev, USSR). *Klin. Khir.* (9):22-29, 1974.

2166 DETERMINATION OF HEME COMPOUNDS IN ACUTE PANCREATITIS. *(Rus.)* Postolov, M. P. (Tashkent Med. Inst., USSR). *Khirurgiia (Mosk.)* (11):81-83, 1973.

See also, 1905, 1906, 1907, 1908, 1909, 2148.

LIVER AND BILIARY TRACT

2167 METASTATIC CARCINOID TO THE LIVER TREATED BY HEPATIC DEARTERIALIZATION. *(E.)* McDermott, Jr., W. V.; Hensle, T. W. (N. Engl. Deaconess Hosp., Boston, Mass.). *Ann. Surg.* 180(3):305-307, 1974.

The effect of hepatic dearterialization on the carcinoid syndrome is described in a 53 yr old female patient, hospitalized for the 4th time with recurrent and progressive carcinoid syndrome. Pre- and postoperative angiography, biochemical parameters, and histopathological findings are presented. This patient had been followed carefully for 4 yr and had been initially treated by hepatic resection, later by chemotherapy and finally by hepatic dearterialization as the tumor regrew and the carcinoid syndrome became incapacitating. Clinical remission was documented by changes in the excretion of 5-hydroxy-indole-acetic acid and by comparison between pre- and postoperative arteriography and by microscopic sections taken at re-exploration for drainage of a necrotic area in the liver. Six months after the dearterialization, the patient had minimal symptoms of occasional flushing and had resumed a normal life. It is pointed out that the technique of ligation is important; proximal ligation of the main hepatic artery should be combined with distal ligation of possible collateral pathways and division of the falciform, coronary and triangular ligaments as well as any obvious branches of the inferior phrenic artery.

2168 PHYSIOPATHOLOGICAL AND CLINICAL SIGNIFICANCE OF HEPATOCYTE STORAGE (S) AND THE TRANSPORT MAXIMUM (Tm) OF BSP IN CIRRHOSIS AND CHRONIC HEPATITIS: CORRELATIONS WITH SINUSOIDAL FLOW. *(It.)* Di Simone, A.; Gentile, S.; Riegler, G.; Coltorti, M. (1st Fac. Med., Univ. Naples, Italy). *Boll. Soc. Ital. Biol. Sper.* 49(23):1411-1414, 1973.

BSP was infused i.v. (0.3 mg/min/kg for 1-3 hr) into 15 patients with cirrhosis, 5 with chronic hepatitis (3 with chronic aggressive and 2 with chronic persistent forms), and 15 normal controls; infusions were continued for 2 more hr at 0.3 and 0.6 times the initial rate, resp. BSP determinations were performed each hour, and S and T_m were calculated by Wheeler's method. Hepatic sinusoidal flow and the plasma clearance constant, K_c, were determined by using the technique of Coltorti *et al.* with a colloidal solution of $^{198}AU\text{-}S_3$. K_c, sinusoidal flow, and T_m were all significantly decreased in cirrhosis. In chronic hepatitis, the values of these parameters were intermediate between those found in cirrhotics and those in controls. Calculation of correlations between the various parameters and partial correlations between T_m and K_c indicates that there is (1) no correlation between the course of T_m and S; (2) a significant correlation between changes in T_m and K_c, which was confirmed by calculation of partial correlations; and (3) no correlation between S and K_c. These findings indicate that changes in BSP clearance in cirrhosis are due primarily to defects in transport and excretion; hepatocyte storage remains essentially unchanged. Apparently hepatocyte storage of BSP is not affected by decreases in T_m and sinusoidal flow.

2169 BILE SALTS AND LIVER DISEASE IN CHILDHOOD. *(E.)* Javitt, N. B. (New York Hosp. - Cornell Univ. Med. Coll., New York, N.Y.) *Postgrad. Med. J.* 50(584):354-361, 1974.

Cholestatic syndromes in the neonate were investigated with respect to newly found bile acid pathways involving conversion of cholesterol directly to monohydroxy bile acids. A 3-month-old child was diagnosed with extrahepatic biliary atresia, and the blood serum bile acid patterns were found to show an extremely high proportion of chenodeoxycholate. Further investigation of these patterns in 25 other cases of infant extrahepatic atresia revealed significantly high proportions of chenodeoxycholate (30-130 g/ml as opposed to normal values of 1-2 g/ml) in all. These patterns correlated much more closely with those of hepatitis than those of other cholestatic syndromes. It is suggested that extrahepatic atresia in infants may be a sequela of neonatal hepatitis rather than a congenital anatomical malformation as previously supposed.

2170 MORPHOLOGICAL INVESTIGATIONS OF PUNCH BIOPSIES OF THE LIVER IN PRECLINICAL WILSON'S DISEASE. *(Ger.)* Mall, G.; Rossner, J. A. (Pathol. Inst., Univ. Heidelberg, Germany). *Verh. Dtsch. Ges. Pathol.* 57:238-243, 1973.

Morphometric examinations were made of liver biopsies from 4 sisters, aged 9-15 yr: 2 had preclinical Wilson's disease and 2 had no evidence of pathological copper deposits in the liver. In preclinical Wilson's disease nonvacuolized lysosomes were decreased and vacuolized lysosomes increased. The increased number of vacuolized "dense bodies" is probably a manifestation of a nonspecific reaction of liver cells to damage. The percentage of the cell volume occupied by the nucleus was somewhat increased. An increase in the number of mitochondria/cell was proportional to the increase in nuclear volume. Mitochondrial volume was increased only in 1 patient who had more marked fatty degeneration and mitochondrial changes. These findings indicate that small mitochondria are found initially in preclinical Wilson's disease, but their volume increases as the disease progresses. It is suggested that the increase in the number of mitochondria and the proportional increase in nuclear volume in more advanced stages of the disease could be regarded as an attempt by the liver to compensate for the damage produced by copper. Large differences in liver cell volume in the 2 normal sisters were caused by differences in the volume of ground substance. This finding was confirmed in 2 follow-up biopsies taken over a 2-yr period and may be caused by recessive Wilson's disease.

2171 THE LIVER IN GM_1 GANGLIOSIDOSIS TYPES 1 AND 2. A LIGHT AND ELECTRON MICROSCOPICAL STUDY. *(E.)* Petrelli, M; Blair, J. D. (Cleveland Metrop. Gen. Hosp., Ohio). *Arch Pathol.* 99(2):111-116, 1975.

The histochemistry and ultrastructure of the liver were studied to attempt to correlate morphological differences between the 2 clinically recognized forms of GM_1 gangliosidosis. Biopsies were obtained from a male infant with the type 1 form of the disease, and a male infant with the type 2 form, characterized by late onset and absence of visceromegaly. Open, live biopsies were performed, and the specimens stained according to Bancroft for light microscopy. For the electron microscopy, specimens were fixed in 4% glutaraldehyde, postfixed in 1% OsO_4 and embedded in epoxy resin. It was found in type 1 that most liver cells contained vacuoles not staining for acid mucopolysaccharides. Most of the Kupffer cells were similarly affected. It was presumed that, since chemical tests had shown a 7-fold increase in liver acid mucopolysaccharides, these substances had been in the vacuoles but had been lost during the histochemical processing. The Kupffer cells were found to contain a granulofibrillar material, identified as a ganglioside by its PAS-positive reaction; however, its ultrastructure was different from that of those found in the brain. In the type 2 specimens, few hepatocytes ha. vacuoles, but the vacuoles that were found had distinctive folded membranes. The Kupffer cells were affected in clusters, had a strong PAS reaction, and the ultrastructure revealed a fine granulofibrillar material. It is concluded that the scarcity of affected cells in type 2 correlates with the normal liver size and acid mucopolysaccharide and ganglioside values. The distinctive appearance and morphological grouping of the Kupffer cells in this type may prove to be of diagnostic importance.

2172 CLINICAL SPECTRUM OF THE BUDD-CHIARI SYNDROME AND ITS SURGICAL MANAGEMENT. *(E.)* Langer, B.; Stone, R. M.; Colapinto, R. F.; Meindok, H.; Phillips, M. J.; Fisher, M. M. (Univ. Toronto, Fac. Med., Canada). *Am. J. Surg.* 129(2):137-145, 1975.

Five patients with Budd-Chiari syndrome were studied with respect to etiology, clinical course, diagnosis, and therapy. Four out of the 5 women studied were on oral contraceptives. Common presenting features were severe abdominal pain, gross ascites and hepatic enlargement. An important aspect of the clinical course pointed out is the presence of a prodromal period between the first mild symptoms and the full-blown disease, which was observed in 4 out of the 5. The histories included anorexia, epigastric discomfort, abdominal swelling, and nausea. Surgery was undertaken in 2 after irreversible thrombosis of the portal vein had occurred, and the shunts clotted with fatal results. In a third patient, after an unsuccessful spleno-venal shunt, a second operation to create a side-to-side mesocaval shunt was carried out with successful results. One patient treated with anticoagulants still required salt restriction and diuretic therapy for ascites even after 10 months, and the hepatic veins slowly continued to narrow. Therefore an early aggressive surgical approach is recommended for treatment in most cases. Diagnosis was made on none during this period, and 2 died due to late discovery of the disease. Hepatic scintiscan, liver biopsy, and most importantly, hepatic venography are cited as useful diagnostic procedures.

2173 LIVER IN PROGRESSIVE MYOCLONUS EPILEPSY (LAFORA'S DISEASE). *(Ger.)* Huchzermeyer, H.; Gerhard, L. (Pathol. Inst., Univ. Dusseldorf, Germany). *Klin. Wochenschr.* 52(12):559-567, 1974.

Histological, histochemical, and clinical studies on progressive myoclonic epilepsy (Lafora's disease) are reviewed, and original observations are presented. A moderate proliferation of the collagenic connective tissue, along with infiltration of lymphocytes, plasmocytes, and leukocytes with polymorphous nuclei between the connective tissue fibers were found in Glisson's fields in Lafora's disease type I after 3 yr. Histochemical investigations revealed intracytoplasmic deposits in hepatocytes primarily of the lobular periphery, diffuse interlobular periseptal fibrosis with reactive interlobular inflammatory infiltration, as well as granular and filamentous components of the hepatocyte deposits. The findings are in good agreement with previous studies.

2174 DEMONSTRATION OF FLUORESCENT, AFLATOXIN B_1-TYPE SUBSTANCES IN THE HUMAN LIVER. *(Fr.)* Richir, C.; Morard, J. L.; Larcebau, S.; Pujol, J. (Fac. Med., Univ. Bordeaux II, Gradignan, France). *Arch. Fr. Mal. App. Dig.* 63(5):391-394, 1974.

Quantitative determinations of aflatoxins were performed on liver tissue, obtained at autopsy, from 50 French patients, aged 24-88 yr (mean age 63 yr), with cirrhosis (11 cases), cardiac liver (10), fatty liver (9), hepatic metastases (4), primary liver cancer (1), hemochromatosis (2), and miscellaneous diseases (8); 5 subjects had normal livers. Residue from a chloroform extract of homogenized liver tissue was first fractionated by chromatography on an alumina column and then identified by thin-layer chromatography on silica gel. Aflatoxin B_1-like material was detected in the livers of 2 patients with alcoholic cirrhosis and portal hypertension and 1 with acute hemorrhagic necrotizing pancreatitis. The aflatoxin contents were 3 µg/kg and 1.6 µg/kg in the 2 cases in which they were large enough to be measured; the liver of the remaining patient contained only traces. These values are much lower than others previously reported in the literature; this might be due to incomplete extraction or to the possibility that some aflatoxin disintegrates after death. The two patients with cirrhosis both drank large quantities of wine, but studies of various commercially available red and white wines failed to disclose the presence of aflatoxin. It is suggested that aflatoxin might be involved in the development of liver cancer in cirrhotics which has been observed in France.

2175 INTRACELLULAR LOCATION OF α-FETOPROTEIN IN LIVER DISEASE. *(Fr.)* Uriel, J. (Inst. Sci. Res. Cancer, Villejuif, France). *Bull. Cancer (Paris)* 60(4):363-370, 1973.

By using ^{3}H-estrone or ^{3}H-estradiol, α-fetoprotein (α-FP) was studied autoradiographically in (1) rat liver cells during histogenesis, (2) hepatocellular carcinoma in man and the rat, and (3) nonneoplastic liver disease. In liver from fetal and newborn rats, α-FP was present in hepatocytes but not in hematopoietic or Kupffer cells or cells of the bile canaliculi. Labeled cells, which never exceeded 1-2% of the entire population, were found in isolation or in small groups, primarily near blood vessels. Within 4-5 days after birth, α-FP also appeared in cells near the portal spaces in areas where bile canaliculi were proliferating. These labeled cells were morphologically intermediate between small hepatocytes and cells covering the bile canaliculi. The cytoplasm or, less frequently, the nucleus and cell membranes of similar cells contained α-FP in primary liver tumors in adult rats and man. In both species these cells were most often found in (1) isolation among the tumor trabeculae, frequently at the edge of capillary sinusoids; (2) grouped near portal spaces in areas where canaliculi were undergoing neoplastic proliferation; and (3) as inclusions in areas of stromal reaction, along with fusiform and proliferating canalicular cells. As in fetal rat liver, a relatively small number of cells secreting α-FP were sufficient to cause high serum titers, as determined by immunodiffusion. In a liver biopsy from a patient with viral hepatitis, α-FP was present in cells similar to labeled cells observed in rats older than 4-5 days and in primary liver tumors. This cell type was also found in the livers of rats which had ingested a diet containing aflatoxin for 2 months. This labeling was observed for 2 months after discontinuation of the carcinogen when no signs of neoplastic transformation were visible. In both cases, labeled cells were near foci of regeneration. Although the exact nature of cells secreting α-FP could not be established, they appear to be immature transitional cells which could either differentiate into hepatocytes or cholangiocytes and which appear to contribute, under some conditions, to hepatic regeneration.

2176 ANALYSIS OF BROMSULPHTHALEIN DISAPPEARANCE CURVES, RELATIVE STORAGE CAPACITY, TRANSPORT MAXIMUM, AND 45-MINUTE DYE RETENTION. *(Ger.)* Spech, H. J.; Wernze, H. (Med. Clin. Univ. Wurzburg, Germany). *Klin. Wochenschr.* 52(10):499-504, 1974.

Relationships between bromosulphthalein elimination, relative storage capacity, transport maximum, and 45-min dye retention were studied in 24 male and female patients with hepatic dysfunctions (cirrhosis, diabetic and alcoholic fatty liver, and liver metastasis in 11, 12, and 1 cases, resp.), and compared with 12 normal controls. Elimination curves obtained following injection of bromosulphthalein (5 mg/kg, i.v.) were compared with relative storage capacity and transport maximum determined by the infusion technique started 60 min after the single injection of the dye. The initial elimination constant k_1 was significantly reduced in patients with both cirrhosis and fatty liver, compared to normal subjects. A considerable percentage of these patients showed values within the lower range of standard variation of normals. The overall elimination constant k_2 was significantly reduced only in the cirrhotic patients. All patients and control subjects showed a significant correlation between k_2 and bromosulphthalein transport maximum at a correlation factor of 0.856, but not between elimination constant k_1 and the relative storage capacity. The 45-min dye retention, used as

an isolated value of the elimination curve, showed a significant correlation with both the elimination constant k_2 and the transport maximum with respective correlation factors of 0.646 and 0.720, but not with the elimination constant k_1. The elimination constant k_2 and the 45-min dye retention over 1.0 mg% are suggested to be quantitative parameters of hepatobiliary bromosulphthalein excretion compared to the more complex measurement of the transport maximum. The relationship between the 45-min dye retention and the transport maximum, though statistically significant, is subject to great variations in individual cases. The findings indicate the absence of uniform, disease-specific bromosulphthalein elimination curves in fatty liver and liver cirrhosis.

2177 A MORPHOMETRIC STUDY OF THE ENDOPLASMIC RETICULUM IN HUMAN HEPATOCYTES. CORRELATION BETWEEN MORPHOLOGICAL AND BIOCHEMICAL DATA IN SUBJECTS UNDER TREATMENT WITH CERTAIN DRUGS. *(E.)* Jezequel, A. M.; Koch, M.; Orlandi, F. (Univ. Ancona Sch. Med., Italy). *Gut* 15(9):737-747, 1974.

A morphometric study was conducted on the endoplasmic reticulum of hepatocytes obtained by needle biopsy from 4 normal, untreated patients and from 4 patients with no liver disease who received diazepam (20-60 mg for 2-4 days), from 1 patient with Gilbert's syndrome who was given phenobarbital (360 mg) for 4 days, and from 1 normal patient who received dilantin (50 mg) for 7 days. In normal, untreated subjects, the amount of smooth endoplasmic reticulum exceeded that of rough endoplasmic reticulum and accounted for 76.3% of the total hepatocyte endoplasmic reticulum. Two components of the smooth endoplasmic reticulum were identified: a vesicular component with a regular honeycomb pattern made up of cisternae with patent lumina and a mean width of 1500 Å (type 1) and a non-vesicular component which occurs in discrete foci of densely packed smooth membranes with a spacing of 140 Å (type 2). Type 1 and type 2 smooth endoplasmic reticulum comprised 2.23 and 0.68 μ^2/μ^3 of cytoplasm, resp., compared to the total smooth endoplasmic reticulum surface density of 2.91 μ^2/μ^3 in normal subjects. Short-term diazepam treatment caused no change in the rough endoplasmic reticulum (0.89 μ^2/μ^3) but did significantly increase the smooth endoplasmic reticulum with a 2- to 3-fold increase of type 2 smooth endoplasmic reticulum in 3 of the 4 patients. ^{14}C-acetate incorporation into digitonin-precipitable sterols (cholesterol) as measured in biopsy materials was increased by about 2.5-15-fold in the same 3 patients compared to normal controls. No changes in endoplasmic reticulum or in cholesterol synthesis were observed in the dilantin-treated patient or in the phenobarbital-treated patients. The increase in the type 2 smooth endoplasmic reticulum membranes follows the administration of substances known to induce microsomal enzymes, and it is suggested that the type 2 represents newly formed while the type 1 represents mature membranes.

2178 PRIMARY CARCINOMA OF THE JUVENILE LIVER: HYPERGAMMAGLOBULINEMIA, α_1-FETOPROTEIN, AND POSITIVE AUSTRALIA ANTIGEN. *(Sp.)* Rodriguez Cuartero, A.; Nunez Carril, J.; Urbano Jimenez, F. (San Cecilio Clin. Hosp., Granada, Spain). *Rev. Esp. Enferm. Apar. Dig.* 44(5):509-520, 1974.

2179 ALPHA-FETOPROTEIN IN PRIMARY LIVER CANCER AND OTHER DISEASES. *(E.)* Kew, M. (Johannesburg Gen. Hosp., South Africa). *Gut* 15(10): 814-821, 1974.

2180 RESECTION OF THE LEFT LOBE OF THE LIVER FOR PRIMARY LIVER CANCER. *(Rus.)* Valter, V. G.; Zurnadzhiants, V. A. (Astrakhan Med. Inst., USSR). *Khirurgiia (Mosk.)* (3):113-114, 1974.

2181 HEPATIC METASTASES: CLINICAL FEATURES, ENDOSCOPY, AND BIOLOGY. *(Ger.)* Hofstetter, J. R.; Realini, S. (Med. Polyclin., Univ. Lausanne, Switzerland). *Z. Gastroenterol.* 12(1): 49, 1974.

2182 HEPATECTOMY FOR LOCALIZED LESIONS. *(Rus.)* Alperovich, B. I. (Tomsk Med. Inst., USSR). *Vestn. Khir.* 113(11):31-34, 1974.

2183 LIVER DISEASE AND PREGNANCY. *(Rus.)* Benediktov, I. I.; Balezin, L. Z. (Sverdlovsk Med. Inst., USSR). *Akush. Ginekol. (Mosk.)* (3): 54-55, 1974.

2184 THE INFLUENCE OF CHRONIC LIVER DISEASE ON THE ELIMINATION OF D-PROPRANOLOL, ANTIPYRINE, AND INDOCYANINE GREEN. *(E.)* Branch, R. A.; James, J. A.; Read, A. E. (R. Infirm., Bristol, England). *Gut* 15(10):837-838, 1974.

2185 STUDIES OF VITAMIN B_{12} ABSORPTION IN CHRONIC LIVER DISEASE. *(Ger.)* Konrad, H.; Kiencke, H.; Teichmann, W.; Bielka, B. (Med. Clin., Univ. Rostock, Germany). *Radiol. Diagn. (Berl.)* 14(6):759, 1973.

2186 CELL-MEDIATED IMMUNITY TO HUMAN TAMM-HORSFALL GLYCOPROTEIN IN AUTOIMMUNE LIVER DISEASE ASSOCIATED WITH RENAL TUBULAR ACIDOSIS. *(E.)* Tsantoulas, D. C.; McFarlane, I. F.; Portmann, B.; Eddleston, A. L. W. F.; Williams, R. (King's Coll. Hosp., London, England). *Gut* 15(10):826, 1974.

2187 THE CLINICAL COURSE OF REYE'S SYNDROME. *(Ger.)* Mutz, I.; Grubbauer, H. M.; Weikmann, E.; Muller, W. (Pediatr. Clin., Univ. Graz, Austria). *Wien. Klin. Wochenschr.* 86(24):745-749, 1974.

2188 ABSCESSES OF THE LIVER. *(Fr.)* Pellet, B.; Ryncki, P. V.; Mir, A.; Mir, M. (Canton. Hosp., Lausanne, Switzerland). *Helv. Chir. Acta* 41(5/6):683-690, 1974.

2189 SURGICAL TREATMENT OF HYDATID CYSTS OF THE LIVER: CONCERNING A SERIES OF 381 CASES. *(Fr.)* Mentouri, B.; Daoud, K.; Haroud, M.; Hammani, A. (Surg. Clin., Univ. Algiers, Algeria). *Helv. Chir. Acta* 41(5/6):675-681, 1974.

2190 LYSOSOMAL CHANGES IN HUMAN AND EXPERIMENTAL IRON OVERLOAD. *(E.)* Seymour, C. A.; Budillon, G.; Peters, T. J. (R. Postgrad. Med. Sch., London, England). *Gut* 15(10):838, 1974.

2191 LIVER FUNCTION IN PATIENTS WITH MUSCULOSKELETAL TRAUMA. *(Rus.)* Eselev, M. M.; Usvitskii, A. E. (Saratov Med. Inst., USSR). *Ortop. Travmatol. Protez.* (7):69-71, 1974.

2192 GASTROINTESTINAL BLEEDING CAUSED BY RUPTURE OF AN ANEURYSM OF THE HEPATIC ARTERY OR ONE OF ITS BRANCHES: ONE CASE REPORT. *(Fr.)* Desligneres, S.; Rouer, K.; Abelanet, R.; Bricaire, H. (Cochin Hosp., Paris, France). *Cah. Med.* 14(10): 883-887, 1973.

2193 HEPATOSPLENIC SYNDROME IN YEMEN. *(Rus.)* Skripnichenko, V. A. (Kiev Med. Inst., USSR). *Vrach. Delo* (12):127-129, 1974.

2194 FULMINANT HEPATIC FAILURE: HISTOLOGICAL QUANTITATION OF SURVIVING LIVER PARENCHYMA IN THE EVALUATION OF THE CAUSE OF DEATH. *(E.)* Portmann, B.; Gazzard, B. G.; Murray-Lyon, I. M.; Williams, R. (King's Coll. Hosp., London, England). *Gut* 15(10):830, 1974.

2195 HEPATIC RESECTION: LITERATURE REVIEW. *(Rus.)* Milonov, O. B.; Starikova, V. B.; Fenomenov, A. M. (All-Union Sci. Res. Inst. Exp. Clin. Surg., Moscow, USSR). *Khirurgiia (Mosk.)* (11):141-146, 1973.

2196 SURGICAL ANATOMY OF THE LIVER: VARIATIONS AND DEVELOPMENT OF TECHNIQUES FOR HEPATECTOMY AND SHUNTS. *(Fr.)* Ryncki, P. V. (Dep. Surg., Univ. Lausanne, Switzerland). *Helv. Chir. Acta* 41(5/6):543-574, 1974.

See also, 1611, 1756, 1813, 1850, 1877, 1888, 1900, 1910, 1911, 1912, 1913, 1914, 1915, 1916, 1917, 1918, 1923, 2131, 2156, 2199, 2222, 2247, 2253, 2257, 2260, 2277, 2290, 2294, 2309.

LIVER AND BILIARY TRACT

Hyperbilirubinemic States

2197 SIGNIFICANCE OF ALBUMIN-BILIRUBIN BINDING IN DRUG THERAPY IN THE NEWBORN. *(Ger.)* Windorfer, Jr., A.; Karitzky, D. (Pediatr. Clin., Univ. Freiburg/Breisgau, Germany). *Monatsschr. Kinderheilk.* 123(1):27-30, 1975.

Sephadex gel filtration was used to study the effect of a variety of drugs on the albumin-bilirubin complex (molar ratio 1:1). When used at maximum concentrations found in the serum of premature and newborn infants (50 and 100 µg/ml), ampicillin, carbenicillin, cephalothin, colistin, erythromycin, gentamicin, penicillin, and phenobarbital displaced little bound bilirubin (0.3-1.3%). When added in concentrations far above those found in premature and newborn sera, digoxin, furosemide, isoproterenol, and triflupromazine displaced 1.5-2.6% of the bound bilirubin. Drugs which have been shown to displace bound bilirubin in premature and newborn infants were added in concentrations found in the serum of these infants. Diazepam, novobiocin, oxacillin, and trimethoprim (all at 50 and 100 µg/ml) and sulfamethoxazole (100 and 200 µg/ml) displaced 2.7% bilirubin or less; a combination of sulfamethoxazole and trimethoprim (50 and 100 µg/ml) displaced 2.5% and 3.5%, resp.; and sodium salicylate (200 and 300 µg/ml) displaced 7.9% and 10.5%, resp. Exchange transfusions of fresh heparinized blood decreased free fatty acid levels and increased albumin-bilirubin binding in 12 cases; increased free fatty acid levels and decreased bilirubin binding in 5; and decreased both free fatty acids and bilirubin binding in 2. Drugs suspected of interfering with bilirubin binding should not be given to newborns with hyperbilirubinemia unless absolutely necessary, and then they should be administered simultaneously with human albumin.

2198 THE EFFECT OF INDOCYANINE GREEN ON ALBUMIN-BILIRUBIN BINDING IN THE NEONATAL PERIOD. *(Ger.)* Kretzschmar, G. W. (Pediatr. Clin., Univ. Stockholm, Sweden). *Monatsschr. Kinderheilk.* 123(1): 31-33, 1975.

Fractionated Sephadex gel filtration was used to compare the effect of indocyanine green on albumin-bilirubin binding with that of sulfafurazole, a drug which is known to cause kernicterus in newborns who have even mild hyperbilirubinemia. Indocyanine green displaced more bilirubin from the albumin-bilirubin complex than did sulfafurazole. However, because of the small doses of indocyanine green employed for dye dilution curves (0.1 mg/kg) and its short half-life, indocyanine green in unlikely to produce kernicterus. The only risk would be in premature infants given exchange transfusions. These transfusions increase the concentrations of free fatty acids which also interfere with bilirubin binding, and the overall effect of these factors might result in kernicterus. In contrast, sulfafurazole is excreted so slowly that plasma concentrations of 150 mg/100 ml can be reached; this is equimolar to a bilirubin concentration of 320 mg/100 ml.

2199 EFFECTS OF CALORIC AND NONCALORIC MATERIALS IN FASTING HYPERBILIRUBINEMIA. *(E.)* Barrett, P. V. D. (Harbor Gen. Hosp., Torrance, Calif.). *Gastroenterology* 68(2):361-369, 1975.

The roles of p.o. and i.v. administered nutrients, and of physical factors were investigated in the reversal of fasting hyperbilirubinemia. Subjects were normal (4), or were patients with chronic aggressive hepatitis (2), Gilbert's syndrome (2), hereditary spherocytosis (2), primary biliary cirrhosis (2), or alcoholism (1), and were fasted for 15 to 39 hr prior to the study. Water, saline, mannitol, glucose, amino acids and medium chain triglycerides were given p.o in equicaloric amounts of 400 cal (mannitol and saline were at a concentration of 1220 mOsm). Mannitol, glucose, and amino acids were also given i.v. at resp. concentrations of 100 g/liter, 100 g/liter, 50 g/liter, resp., and saline at 301 mEq/liter, An *in vitro* study was also performed in which normal saline, glucose (100, 200 or 400 mg/100 ml) or mannitol (400 mg/100 ml) was incubated with whole blood from an icteric patient to rule out possible influences of the diazo reaction on serum bilirubin determination. It was found that p.o. administration of water caused an increase in the serum bilirubin, while saline, mannitol, amino acids, and triglycerides did not alter it. In contrast, glucose caused a significant decrease of 8% within 3 hr. I.v. glucose, mannitol, and amino acids caused a significant increase similar to that of the p.o. water (21-61% over control values). I.v. or p.o. loading with hypertonic saline, osmotically equivalent with the i.v. glucose and mannitol concentrations, failed to produce an increase in total serum bilirubin, ruling out osmotic factors. It is concluded that glucose is the factor responsible for reversal of fasting hyperbilirubinemia after eating.

2200 DUBIN-JOHNSON SYNDROME AND RELATED SYNDROMES: THEIR CLASSIFICATION. *(It.)* Coppo, M. (Inst. Med. Clin., Univ. Modena, Italy). *Fegato* 19(2):469-479, 1973.

Rotor's syndrome and Burka's syndrome are generally considered related to Dubin-Johnson syndrome. In Rotor's syndrome the patients are jaundiced but the liver is not pigmented, while in Burka's syndrome the liver is pigmented but the patient is anicteric. According to his experience, the author considers this classification arbitrary and unacceptable. In Dubin-Johnson syndrome the characteristic pigmentation often develops after the onset of jaundice, and the chronology of functional defects in hepatocyte excretion differs from one case to another. Dubin-Johnson syndrome cannot be ruled out if the liver is a mottled green, rather than black, at laparoscopy. It may be that Rotor's syndrome is actually a mild, initial, or regressive form of Dubin-Johnson syndrome. Intercurrent acute hepatitis sometimes results in chronic hyperbilirubinemia followed by complete loss of hepatic pigmentation, so it may appear that Dubin-Johnson syndrome has been transformed into Rotor's syndrome. In contrast, Dubin-Johnson syndrome and Burka's syndrome are believed to be distinct clinical entities. It has been demonstrated that results of the BSP clearance test and the ultrastructure of hepatic pigmentation are quite different in these 2 syndromes. Dubin-Johnson syndrome is defined as a dissociated form of intrahepatic cholestasis in which conjugated bilirubin appears in the blood under basal conditions and after i.v. injection of free bilirubin; conjugated BSP appears in the blood after i.v. injection of free BSP; and indocyanine green clearance is normal. Patients have no pruritus, bile acid retention, hypercholesteremia, or increased serum alkaline phosphatase levels.

2201 HISTOPATHOLOGICAL, HISTOCHEMICAL, AND ULTRASTRUCTURAL STUDIES OF THE LIVER IN SOME PATIENTS WITH DUBIN-JOHNSON AND ROTOR'S SYNDROMES. *(It.)* Sciacca, A.; Soccorsi, P.; di Giacomo, V.; Carmenini, G.; Fragola, P. (Inst. 1st Med. Clin., Univ. Rome, Italy). *Fegato* 19(2):499-531, 1973.

From an extensive review of the literature and their own findings, the authors conclude that there is no unequivocal evidence to demonstrate that transport disorders in Dubin-Johnson and Rotor's syndrome are the result of ultrastructural lesions in the liver. The authors have rarely found mitochondrial changes, and abnormalities in the endoplasmic reticulum and vascular pole of the hepatocytes were later identified as artifacts. The significance of dilatations in intercellular space is unknown, but this change has been observed in a variety of diseases accompanied by liver cell damage. No relation could be established between the rare finding of "curly" structures and the disorder in bilirubin excretion. Studies of liver pigmentation in Dubin-Johnson syndrome suggest that the large deposits of pigment consist of a mixture of lipofuscin and melanin, while the smaller deposits present in some patients with Rotor's syndrome consist primarily of lipofuscin.

2202 INTRAHEPATIC STORAGE CURVE FOR ^{3}H-BILIRUBIN IN GILBERT'S DISEASE BEFORE AND AFTER PHENOBARBITAL THERAPY. *(It.)* Tiribelli, C.; Cobelli, C.; Frezza, M.; de Sandre, G. (Inst. Spec. Med. Pathol., Univ. Trieste, Italy). *Fegato* 19(2):423, 428, 1973.

Curves for intrahepatic storage of ^{3}H-bilirubin were obtained from curves for plasma clearance of ^{3}H-bilirubin plotted for 3 normal subjects and 6 patients with Gilbert's disease before and after phenobarbital therapy (150 mg/day for 7 days). Before treatment, patients with Gilbert's disease had significant increases in 2 of the 4 transfer constants calculated. These were the storage time (ta), the time needed to pass from 90% to 90% of the maximum value in the 2 slopes of the curve, and the decrease time (td), the time needed to decrease from the maximum value to 50% of the maximum. The relative height of the intrahepatic storage curve, dpm to/tp, was also significantly increased in patients with Gilbert's disease. After phenobarbital treatment, the transport constants remained essentially unchanged, while the relative height of the intrahepatic ^{3}H-bilirubin storage curve decreased to normal values. The in-

creases in transfer constants in Gilbert's disease are attributed to a decrease in bilirubin storage in the liver and to disorders in excretion which may be due to failure of bilirubin to conjugate with glucuronic acid.

2203 STEREOMETRIC STUDY OF THE OCCURRENCE OF GIANT LIVER MITOCHONDRIA IN PATIENTS WITH GILBERT'S DISEASE AS COMPARED TO NORMAL CONTROLS. *(Fr.)* Slabodsky-Brousse, N.; Feldmann, G.; Brousse, J.; Dreyfus, P. (Beaujon Hosp., Clichy, France). *Biol. Gastroenterol. (Paris)* 7:179-186, 1974.

Ultrastructural stereometric techniques were used to determine the relative volume, relative surface area, and ratio of surface area to volume of liver mitochondria in 9 patients with Gilbert's disease (7 men and 2 women, aged 18-45 yr) and 10 controls (6 men and 4 women, aged 20-60 yr) with no clinical, biological, or histological evidence of liver disease. None of the patients or controls was a chronic alcoholic or had received estrogens. No difference between these 2 groups was found for the mean relative volume occupied by mitochondria (17.93% in Gilbert's disease, 17.03% in controls) or for the mean relative surface area (0.96 $\mu^2:\mu^3$ hepatocyte in Gilbert's disease, 1.03 $\mu^2:\mu^3$ hepatocyte in controls). The mean ratio of surface area:volume of the mitochondria was 5.34 $\mu^2:\mu^3$ in Gilbert's disease and 6.13 $\mu^2:\mu^3$ in controls. Mitochondria were then grouped into 39 classes, according to mitochondrial diameters: group I included classes 1 and 2 (0.5-1 μ); group II included classes 3, 4, and 5 (1.5-2.5 μ); and group III included classes 6 through 39 (equal to or greater than 3 μ). Group I mitochondria were significantly more common among controls, while group III mitochondria were significantly more common in patients with Gilbert's disease. The controls had no mitochondria with diameters larger than 4.5 μ. These findings suggest that giant mitochondria are an ultrastructural characteristic of Gilbert's disease. The significance of these giant mitochondria is unknown.

2204 PROLONGED SURVIVAL IN THE CRIGLER-NAJJAR SYNDROME: ULTRASTRUCTURAL AND DIETARY STUDIES. *(E.)* Gollan, J. L.; Huang, S. N.; Billing, B. H.; Sherlock, S. (R. Free Hosp., London, England). *Gut* 15(10):838, 1974.

2205 BILIRUBINOPATHY AND HEREDITARY HEPATOCYTIC ENZYME DEFICIENCY. *(It.)* Giordano, A.; Coggi, G.; Grigolato, P. (Inst. Anat., Univ. Milan, Italy). *Fegato* 19(2):321-331, 1973.

See also, 1773, 1775.

LIVER AND BILIARY TRACT

Nonviral Hepatitis and Hepatic Injury

2206 DRUG METABOLISM IN NORMAL CHILDREN, LEAD-POISONED CHILDREN, AND NORMAL ADULTS. *(E.)* Alvares, A. P.; Kapelner, S.; Sassa, S.; Kappas, A. (Rockefeller Univ., New York, N.Y.). *Clin. Pharmacol. Ther.* 17(2):179-183, 1975.

The metabolism rates of phenylbutazone and antipyrine in children and adults were determined in a study on age dependence of hepatic drug-metabolizing enzymes in man. Ten normal adults (25-40-yr-old) and 10 children (1-8-yr-old) were studied. Of the children, 2 were normal and 8 had biochemical evidence of lead poisoning. Phenylbutazone was administered (6 mg/kg, p.o.), then blood was drawn on days 1-5, and 7 for determination of plasma phenylbutazone levels. Antipyrine dissolved in syrup was administered (18 mg/kg, p.o.) and blood drawn 3, 6, 9, 12, and 15 hr following administration for determination of plasma antipyrine levels. The mean phenylbutazone half-lives in the children and adults (1.68 and 3.16 days, resp.) differed significantly. The mean antipyrine half-life in the whole group of 10 children, 6.63 hr, was significantly lower than the mean half-life of 13.58 hr obtained in adults. There were no differences between the 2 normal children and the 8 lead-poisoned children in their capacities to metabolize the 2 drugs. It appeared that children in the age range studied metabolize drugs at almost twice the rate of adults, which differs from findings in animals in which drug-metabolizing capacities increase with maturation. The results, therefore, indicate that the age dependence of hepatic drug-metabolizing enzymes found in experimental animals may not apply to man, and the increased metabolism of the 2 test drugs observed in children has considerable significance in the calculation of proper dosages of drugs.

2207 SEVERE HEPATITIS FROM METHYLDOPA. *(E.)* Maddrey, W. C.; Boitnott, J. K. (Johns Hopkins Med. Inst., Baltimore, Md.). *Gastroenterology* 68(2):351-360, 1975.

Hepatitis secondary to methyldopa therapy was studied to emphasize the importance of checking for a possible drug etiology in all patients with a histological picture suggesting chronic aggressive hepatitis. Clinical, pathological, and serological studies were made on 6 patients with severe hepatitis following methyldopa therapy. All 6 patients (women, 38-62-yr-old) continued to take methyldopa after the onset of symptoms, and all had laboratory evidence of hepatitis with elevations of serum transaminases. The drug was inadvertently readministered

to 1 patient with recurrence of hepatitis within 1 week; all rapidly improved after discontinuation of the drug. The authors emphasize the need to consider the diagnosis of hepatitis and to determine serum transaminases whenever a patient on methyldopa develops symptoms of fatigue, anorexia, nausea and vomiting; and, also, to discontinue the drug in those patients with abnormal liver function tests and constitutional symptoms suggestive of hepatitis.

2208 QUINIDINE-INDUCED GRANULOMATOUS HEPATITIS. *(E.)* Chajek, T.; Lehrer, B.; Geltner, D.; Levij, I. S. (Hadassah Univ. Hosp., Jerusalem, Israel). *Ann. Intern. Med.* 81(6):774-776, 1974.

A case of quinidine-induced granulomatous hepatitis is reported in a 69-yr-old male. The patient was readmitted with fever of 1 day duration 2 days after having been discharged from another hospital where he had been treated for congestive heart failure with digoxin, furosemide and quinidine. This represented the 1st time he had been treated with quinidine. On physical examination, the patient exhibited bilateral axillary adenopathy and a liver palpable 2 cm below the costal margin. Erythrocyte sedimentation rate was 30 mm/hr (Westergren); WBC, 5200/mm; serum bilirubin, 1 mg%; alkaline phosphatase, 205 mU (normal, 25-85); and SGOT, 430 U (norlam, 10-50). These improved following quinidine withdrawal, but symptoms, physical findings and abnormal liver function tests again returned upon challenge with quinidine. Liver biopsy 5 days after challenge showed numerous granulomas without caseation or giant cells. The patient's indices again returned to normal 1 day after quinidine was discontinued. A 2nd quinidine challenge 2 months later produced the same results as the 1st challenge. This represents the 1st reported case of histologically confirmed quinidine-induced granulomatous hepatitis.

2209 PENICILLIN AND EPLUCHAGE TREATMENT OF HEPATIC ACTINOMYCOSIS. *(E.)* Prioleau, P. G.; Brochu, F. L. (VA Hosp., Salem, Va.). *Arch. Surg.* 109(3):426-429, 1974.

A rare case of primary hepatic actinomycosis is reported in which prolonged penicillin therapy and removal of the involved tissue were successfully employed. A 44-yr-old white male hospital dietary storeroom worker was admitted with a 2-month history of persistent right upper quadrant abdominal pain. This began after he had suffered a traumatic fraction of the right 7th rib, which healed normally. He was a severe alcoholic and had previously undergone surgery for chronic pancreatitis; he had also had an appendectomy and a cholecystectomy. Exploratory laparotomy revealed actinomycosis of the anterior right lobe of the liver. Prolonged penicillin therapy and removal of the involved hepatic tissue in multiple-staged operative procedures were successful in eliminating the disease. The patient died 27 months after diagnosis following prolonged alcoholic ingestion. He had no evidence of active actinomycosis when last examined 10 weeks prior to death. Death from abdominal actinomycosis may be secondary to direct extension of the disease to the lung, kidney, or liver; the cause of death in the present case is unknown.

2210 HEPATITIS CAUSED BY ACQUIRED TOXOPLASMOSIS: TWO CASE REPORTS. *(Fr.)* Beauvais, P.; Koenig, H.; Alexandrovicius, H.; Dorr, R.; Somolo, P. (Mulhouse Hosp. Cent., France). *Pediatrie* 29 (6):615-625, 1974.

2211 AN ELECTRON MICROSCOPIC STUDY OF LIVER IN HEPATOMEGALY PRESUMABLY CAUSED BY AMEBIASIS. *(E.)* Tandon, B. N.; Tandon, H. D.; Puri, B. K. (All-India Inst. Med. Sci., New Delhi). *Exp. Mol. Pathol.* 22(1):118-132, 1975.

LIVER AND BILIARY TRACT

Acute Viral Hepatitis

2212 CONCENTRATION OF HEPATITIS-B ANTIGEN FROM CLINICAL PREPARATIONS OF II, VII, IX, AND X BY POLYETHYLENE GLYCOL, TO ENHANCE DETECTION AND REMOVAL. *(E.)* Johnson, A. J. (New York Univ. Med. Cent., N.Y.). *Ann. N.Y. Acad. Sci.* 240:162-164, 1975.

Hepatitis B antigen (HB Ag) was concentrated from clinical preparations of factor II, VII, IX, and X concentrates by selective precipitation with polyethylene glycol (PEG). Of 27 batches of concentrate intended for clinical use, measurable amounts of HB Ag were found in 70% using the PEG method. In order to assess the efficacy of this procedure, small amounts of antigen-positive serum were added to the concentrates and removed by the PEG method. As shown by radioimmunoassay results, virtually all antigen was removed in the 1st precipitation. After a 2nd fractionation, the remaining antigen was less than 1 standard deviation from the mean of the negative control. This method increased the sensitivity for detecting HB Ag and the assayable antigen was removed before clinical use.

2213 INTRANUCLEAR VIRUS-LIKE PARTICLES IN HEPATOCYTES OF AN ASYMPTOMATIC HEPATITIS B ANTIGEN CARRIER WITH DANE PARTICLES IN THE SERUM. *(E.)* Yamada, G.; Kosaka, K. (Okayama Univ. Med.

Sch., Okayama, Japan). *Gastroenterology* 68(2):370-373, 1975.

A case history is reported for an asymptomatic hepatitis B antigen (HB Ag) carrier (male, 33 yr old) with Dane particles in the serum and many intranuclear virus-like particles within the hepatocytes. Physical examination, blood picture, and liver function tests were normal, except for the indocyanine green excretion test, with a clearance of 0.16. Dane particles with small particles (20 nm) and tubular forms were observed in the serum by electron microscopy. With light microscopy, the liver showed slightly swollen hepatocytes, periportal fibrosis, and eosinophilic granular inclusions in the nuclei of some hepatocytes. Under electron microscopy, many spherical particles measuring 10-25 nm in diameter were found randomly distributed and in clusters in the nuclei of hepatocytes. Many of these particles had a denser peripheral wall, a few were uniformly dense, and the contour of some were hexagonal. Some virus-like particles were seen near the pores of nuclear membranes. It has been suggested that these intranuclear particles and the inner bodies of Dane particles are the same, but there is also the possibility that they are entirely different. These intranuclear virus-like particles may be continuously replicated and appear as Dane particles in the serum of asymptomatic HB Ag carriers.

2214 PATTERN OF CORE AND SURFACE EXPRESSION IN LIVER TISSUE REFLECTS STATE OF SPECIFIC IMMUNE RESPONSE IN HEPATITIS B. *(E.)* Gudat, F.; Bianchi, L.; Sonnabend, W.; Thiel, G.; Aenishaenslin, W.; Stalder, G. A. (Dep. Pathol., Univ. Basel, Switzerland). *Lab. Invest.* 32(1):1-9, 1975.

A series of 67 liver biopsies (20 kidney transplant recipients, immunosuppressed; and 47 outpatients with hepatitis, no immunosuppression) was studied for the presence of hepatitis B antigen core (HB_C) and surface (HB_S) components by immunofluorescence and electron microscopy. HB_C was demonstrated mainly in the hepatocyte nuclei, and HB_S exclusively within the cytoplasm. Based on these findings and histological and clinical parameters, 4 reaction types were identified. Type I (Elimination Type) showed only occasional or no expression of HB_C, was a classic lobular hepatitis, histologically, and clinically an acute, self-limiting viral hepatitis. Type II (HB_C Predominance, or Immunosuppression Type) had HB_C in every hepatocyte nucleus and focal expression of HB_S, showed nonaggressive inflammation (nonspecific reactive or portal hepatitis) histologically, and clinically was a mild, chronic and persistent hepatitis in transplant patients. Type III (HB_S Predominance, or Nonaggressive Type) had prominent HB_S expression with little HB_C; histologically there was nonaggressive inflammation or normal liver tissue, together with ground-glass hepatocytes (as seen with light microscopy correlating with the HB_S-containing hepatocytes), and clinically there was chronic persistent hepatitis and the patient was a HB antigen carrier. Type IV (HB_{C+S} Equivalence, or Aggressive Type) showed occasional expression of both components, especially the core; had periportal hepatitis, histologically; and clinically was a chronic aggressive hepatitis or an acute hepatitis with possible transition to chronicity. It was suggested that immune responsiveness determines the reaction pattern, with immune elimination of affected cells being the key mechanism. The demonstration of HB Ag in hepatocytes of seropositive patients might be a sign of persistent HB virus infection and an indication of an impaired immune elimination of infected cells. These 4 types of reactions are of prognostic value.

2215 DETECTION OF AUSTRALIA ANTIGEN IN URINE AND SALIVA. *(E.)* Tanno, H.; Fay, O. H.; Roncoroni, M.; Garcia, C.; Katz, R. (Parque Sanit., Rosario, Argentina). *Acta Hepatogastroenterol. (Stuttg.)* 21(5):373-376, 1974.

The presence of the Australia antigen (HB_SAg) was studied in the urine and saliva of 13 seropositive patients (6 with acute hepatitis, 4 with chronic hepatitis, and 3 with postnecrotic cirrhosis). HB_SAG was detected in the urine and saliva using counterelectrophoresis. The antigen was found both in the urine and the saliva of 85% (11 of 13) of those tested (in 5 of 6 cases of acute hepatitis, in 3 of 4 cases of chronic hepatitis, and in 3 of 3 cases of postnecrotic cirrhosis). There was no difference found in the types of proteins filtered in the urine between the HB_SAg-positive and the HB_SAg-negative specimens. The presence of the antigen in these body fluids may be important as a means of transmission.

2216 HEPATITIS B ANTIGEN IN SALIVA, URINE, AND STOOL. *(E.)* Irwin, G. R.; Allen, A. M.; Bancroft, W. H.; Karwacki, J. J.; Brown, H. L.; Pinkerton, R. H.; Willhight, M.; Top, Jr., F. H. (Walter Reed Army Inst. Res., Washington, D.C.). *Infect. Immun.* 11(1):142-145, 1974.

A survey of hepatitis B patients, asymptomatic hepatitis B surface antigen (HB_SAg) carriers and control subjects was conducted to d termine the relation between antigenemia and antigen excretion in saliva, urine and stool. From 5-10 ml of saliva and 100 ml of first-morning urine were collected twice weekly. A stool specimen was obtained once a week. Blood was drawn from all subjects. Radioimmunoassay was used to detect HB_SAg in concentrated urine and stool specimens, and unconcentrated saliva and serum samples. Radioimmunoassay-positive sera was tested for specificity by an inhibition test using antibody to surface antigen. Specificity-confirmed HB_SAg was detected in the saliva of 6 of 20 antigenemic patients (30%), in 1 of 20 nonantigenemic patients (5%), in 14 of 41 carriers (34%), and in none of the 112 controls. HB_SAg was detected in urine only after 100-fold concentration. Specificity-confirmed HB_SAg was present in the urine of 7 of 43 carriers (16%) unconfirmed HB_SAg was found in the urine of 5 of 38 patients (13%), and in 5 controls (5%). Unconfirmed HB_SAg was detected in stool specimens from 5 of 11 patients (46%), and in none of the 8 carriers and controls. The findings suggest that when HB_SAg is present in the serum, it may also be in the saliva, urine, and stool. Whether the presence of HB_SAg in these substances will correlate with their infectivity remains to be determined. Until infectivity

tests are possible, however, saliva, urine, and stool from antigenemic persons should be considered infectious.

2217 CLINICAL AND EPIDEMIOLOGICAL SIGNIFICANCE OF THE HB_SAg (AUSTRALIA ANTIGEN): CARRIER STATE. *(E.)* Feinman, S. V.; Cooter, N.; Sinclair, J. C.; Wrobel, D. M.; Berris, B. (Mt. Sinai Hosp., Univ. Toronto, Canada). *Gastroenterology* 68(1): 113-120, 1975.

The clinical and epidemiological significance of the Australia antigen (HB_SAg) was studied up to 30 months in 115 asymptomatic carriers of the antigen in Toronto, Canada. Only 1 carrier had any previous history of icteric disease. There was a male to female ratio of 3.6:1 and the majority of carriers were less than 30 yr old. A high prevalence of carriers was found in persons from Mediterranean countries and the Orient. A moderate elevation of IgG was found in 6 of 77 carriers. No general defect in cell-mediated immunity was found, and the different subtypes of HB_SAg correlated with ethnic origin. Normal liver function tests were found in 53% of the carriers; SGPT was elevated in 30%; thymol turbidity was abnormal in 22%; and BSP retention test was abnormal in 14 of 51 carriers (27%). Features of chronic benign persistent hepatitis were present in 20 of 29 liver biopsies, but there was no transition to aggressive hepatitis during the 30 months of observation. SGPT was the most common abnormal value and this correlated best with biopsy findings. It was shown that age, sex, and ethnic origin were important host factors that influenced the development of the carrier state. All carriers remained well during the follow-up period.

2218 RELATION OF PROTRACTION OF ACUTE VIRAL HEPATITIS TO AUSTRALIA ANTIGEN. *(E.)* Kamada, T.; Akeyama, T.; Noyama, T.; Abe, H. (Osaka Univ. Med. Sch., Japan). *Acta Hepatogastroenterol. (Stuttg.)* 21(5):358-363, 1974.

The role of Australia antigen HB_SAg (as determined by counterimmunoelectrophoresis and single radial immunodiffusion) in the protraction of acute viral hepatitis was studied. The study included 127 acute viral hepatitis patients (86 with no history of transfusion, and 41 with posttransfusion hepatitis; 69 males and 58 females). Diagnosis was made by physical and laboratory findings (including serum bilirubin and transaminases). A protracted case was defined as one where the abnormal laboratory findings extended beyond 180 days. Acute hepatitis was protracted in 29 (22.8%) of the 127 patients. HB_SAg or antibody was detected in 47 (37%) of the 127 and the disease was protracted in 12 (25.6%) of the 47. It was protracted in 17 (21.3%) of 80 antigen-negative patients: thus there was no difference in protraction rate between antigen-positive and negative patients (contrary to some previous studies). The rate of protraction was higher in patients older than 30, and in males that were HB_SAg-positive. In the nontransfusion group the rate of protraction was lower in antigen-negative patients (5.2%) and 25.5% in the positive patients. The disease was protracted in all 7 patients who remained HB_SAg-positive for more than 6 months. This may indicate that HB_SAg plays an important role in the protraction of hepatitis.

2219 SERIAL STUDIES OF HEPATITIS-ASSOCIATED ANTIGEN AND ANTIBODY IN PATIENTS RECEIVING ANTITUMOR CHEMOTHERAPY FOR MYELOPROLIFERATIVE AND LYMPHOPROLIFERATIVE DISORDERS. *(E.)* Wands, J. R.; Chura, C. M.; Roll, F. J.; Maddrey, W. C. (Johns Hopkins Univ. Sch. Med., Baltimore, Md.). *Gastroenterology* 68(1):105-112, 1975.

A population of 85 patients (25 with myeloproliferative disorders and 60 with lymphoproliferative disorders) was serially studied to determine the effects of antitumor chemotherapeutic agents (especially cyclophosphamide, 50 mg/kg/day; and cytosine arabinoside, 50 mg/kg/day) on hepatitis B antigen (HB Ag) and hepatitis B antibody (HB Ab). During the study HB Ag, as determined by hemagglutination inhibition and hemagglutination, was detected in 17 patients and HB Ab in 40 (a titer of 1:4 or greater considered positive). In 17 chemotherapy patients there was a decrease in preexisting HB Ab titers which corresponded to the period of maximal bone marrow suppression. In 14 of the 17, the values returned to pretreatment values after treatment. Five patients with preexisting HB Ab developed HB Ag and a rise in serum transaminase enzymes, suggesting hepatocellular damage during chemotherapy. In 3 of these a reappearance of HB Ab was associated with the disappearance of HB Ag. HB Ag persisted longer than 3 months in the other 2 patients. In 3 patients with pretreatment HB Ag, this titer increased with chemotherapy, parallel with the maximum period of bone marrow suppression. Hepatocellular damage also occurred. In 2 of these patients, the HB Ag titers returned to pretreatment levels. Chemotherapeutic agents caused a decrease in preexisting HB Ab titers and an increase in preexisting HB Ag. Whether this appearance or increase of HB Ag was related to an inhibition of antibody formation, allowing expression of preexisting antigen, or to an inhibition of cellular immunity, allowing viral proliferation, is uncertain.

2220 SERUM LIPOPROTEINS IN ACUTE VIRAL HEPATITIS. *(Ger.)* Baumgarten, M.; Wienbeck, M.; Englhardt, A.; Martini, G. A. (Med. Clin., Univ. Marburg, Germany). *Klin. Wochenschr.* 52(13):617-623, 1974.

Lipoproteins of 20 patients with acute viral hepatitis were studied in native serum and in serum after precipitative ultracentrifugation by means of lipid electrophoresis and immunological methods. In addition, high-density lipid (HDL) fractions were analyzed by disc electrophoresis. The observations covered 1 to 8 weeks. The onset of the disease was counted from the appearance of jaundice. The α-lipoproteins were markedly reduced and continued to decrease further during the first 3 weeks of the disease, while the β-lipoproteins were significantly elevated and continued to rise during the first 3 weeks. The pre-β-lipoproteins were moderately but regularly reduced

during the whole course of the disease. The lipoprotein levels began to normalize 6 weeks after the onset of the disease. Incomplete separation of β- and pre-β-lipoproteins, of α-lipoproteins, and of the lipoproteins within the albumin fraction on the acetate foils was observed in certain patients. However, ultracentrifugal separation indicated that these lipoproteins belong to the well-known fractions, thus indicating an altered speed of migration in the electrical field. In conclusion, all 3 types of α-lipoproteins were detected in the α-lipoproteins of the HDL fraction and typical quantitative and qualitative changes in the serum lipoproteins in the course of acute viral hepatitis.

2221 PLASMA α-LIPOPROTEIN PATTERN IN ACUTE VIRAL HEPATITIS. *(E.)* Thalassinos, N.; Hatziomannou, J.; Scliros, P.; Kanaghinis, T.; Anastasiou, C.; Crocos, P.; Thomopoulos, D.; Gardikas, C. (Evangelismos Hosp., Athens, Greece). *Am. J. Dig. Dis.* 20(2):148-155, 1975.

A detailed study was done to illustrate the serial changes in plasma lipoprotein, triglyceride, and cholesterol levels during the course of acute viral hepatitis. A group of 57 patients (mean age 30.8 yr) with the disease were followed both clinically and biochemically. Blood samples were taken at 10-day intervals to determine serum bilirubin, SGOT, SGPT, plasma lipoprotein, cholesterol and triglycerides. The tests were continued until serum bilirubin was less than 2.5 mg/100 ml. Mean levels of plasma triglyceride were elevated at the onset of the disease and gradually returned to normal; mean plasma cholesterol values gradually increased. α-Lipoprotein was absent at the early stage of hepatitis in 41 of 46 patients and reappeared gradually during the course of the disease. In 11 cases of viral hepatitis with impending or overt coma, α-lipoprotein was absent for the duration of the acute stage and never reappeared in those who eventually died, while it eventually returned to normal in those who survived. This study demonstrates that plasma α-lipoprotein is dramatically reduced during the acute phase of acute viral hepatitis and reappears in ordinary cases within 20-30 days from onset of jaundice; its reappearance appears to be a sensitive index of improvement and a significant prognostic sign.

2222 RELATIONSHIP OF SERUM α-FETOPROTEIN TO THE SEVERITY AND DURATION OF ILLNESS IN PATIENTS WITH VIRAL HEPATITIS. *(E.)* Bloomer, J. R.; Waldmann, T. A.; McIntire, K. R.; Klatskin, G. (Yale Univ. Sch. Med., New Haven, Conn.). *Gastroenterology* 68(2):342-350, 1975.

Levels of serum α-fetoprotein in 176 patients with viral hepatitis were studied in relation to the severity and duration of illness. A radioimmunoassay procedure with a lower limit of sensitivity of 5 ng/ml, was used, and values greater than 40 ng/ml were considered to be increased, as compared to controls. Increased levels of α-fetoprotein were found in 38% of the 176 patients. Of 75 patients with subacute hepatic necrosis (characterized by zones of necrosis that bridged adjacent portal triads and/or central veins and showed more extensive hepatocellular necrosis than classical hepatitis), 52% had increased α-fetoprotein and 9 of these exceeded 500 ng/ml. Only 28% of the 101 patients with classic hepatitis lesions had elevated values, and none of these exceeded 500 ng/ml. A higher percentage of patients under age 30 had increased levels. There was no linear relationship between serum α-fetoprotein and levels of SGOT, serum bilirubin, serum alkaline phosphatase, prothrombin time, and serum proteins. In subacute hepatic necrosis, the highest increase of α-fetoprotein occurred when the symptoms had been present for about 4 weeks, and when levels of SGOT were decreasing. This indicated that the α-fetoprotein peaked when the necrotic phase of the lesion was subsiding. This rise was related to a longer survival time and associated with prominent features of regeneration. The monitoring of the fetoprotein may be an indicator of hepatic regeneration.

2223 A SECOND ANTIGEN-ANTIBODY SYSTEM IN VIRAL HEPATITIS. *(Fr.)* Lemaire, A. (no affil.). *Nouv. Presse Med.* 3(27):1715-1716, 1974.

Research done by Hoofnagle *et al.* on core (nucleocapsid) antigen (CAg) and antibody (CAb) in 350 subjects (patients recovering from hepatitis, blood donors, and hemophiliacs) is reviewed. Pure nucleocapsids were isolated from Dane particles by ultracentrifugation of liver homogenate obtained from a chimpanzee which died after infection with human serum containing Australia antigen (HB_sAg), subtype *ayw*. Radioimmunoassay showed the absence of any protein from the viral mantle, i.e. HB_sAg. Injection of CAg into guinea pigs induced production of antobodies which fixed complement in the presence of CAg, but not in the presence of HB_sAg or liver extract. Studies in humans demonstrated that CAg is specific for hepatitis B: it was never detected in sera from patients with hepatitis A. CAb could be detected 10-20 weeks after contact with an infected subject, i.e. 3 weeks after development of jaundice, and 4-10 weeks after serological detection of HB_sAg. CAb always appeared before Australia antigenies (HB_sAb) when virus was present in the blood and when liver function tests were most abnormal, whether or not the patient had jaundice. CAb occurred more frequently than HB_sAb and was sometimes present in low titers for 3 yr. Although HB_sAb was detected in only 9 chronic carriers of hepatitis, CAb was present in all 50 carriers studied. CAb was found more often in commercial than in volunteer blood donors, but it was less common in both of these groups than HB_sAb, suggesting that it is less persistent than HB_sAb. CAb was less common and was present in lower titers among hemophiliacs than among commercial donors. Since CAb is associated with the persistence of HB_sAg in the circulation, these antibodies may form in response to active replication of the virus. There is as yet no satisfactory explanation for the presence of HB_sAg, HB_s-Ab, and CAb alone or simultaneously in the same serum. These investigations suggest that blood containing high titers of CAg should not be used for transfusion, even if HB_sAg cannot be detected.

2224 POTENCY OF FRAGMENTED IgG: TWO STUDIES OF POSTEXPOSURE PROPHYLAXIS IN TYPE A HEPATITIS. *(E.)* Brachott, D.; Lifschitz, I.; Mosley, J. W.; Kendrick, M. A.; Sgouris, J. T. (Minist. Health, Ramalah, Israel). *J. Lab. Clin. Med.* 85(2): 281-286, 1975.

Studies were performed to determine the effect of fragmentation on potency of immunoglobulin preparations. Immunoglobulin preparations (1 from U.S., 1 from Israel) were compared, in both fragmented and unfragmented forms, with each other and with a placebo. In each of the 2 studies, 3 materials were given to household contacts (2-9 yr of age) of icteric hepatitis: human albumin as a placebo; immunoglobulin with the IgG intact; and immunoglobulin of the same lot with the IgG deliberately fragmented by added fibrinolysin. The dose of globulin administered averaged 0.026 ml (4.3 mg of IgG protein)/kg body weight; the children were observed over a 60-day period. In the 1st study of 1767 children, the number of cases of secondary attack were: 28 of 593 following placebo; 16 of 573 following administration of unfragmented globulin; and 13 of 601 following administration of fragmented globulin. In the 2nd study of 1419 children, the number of cases of secondary attack were: 35 of 463 following placebo; 16 of 479 following unfragmented globulin; and 14 of 477 following fragmented globulin. The results showed that comparable reductions in secondary attack rates were achieved with fragmented and unfragmented materials from both lots of globulin. The authors conclude that fragmentation by fibrinolysin does not lower potency for postexposure prophylaxis of hepatitis A.

2225 NEW CONTRIBUTION TO THE PROBLEM OF CONNATAL VIRAL HEPATITIS. *(E.)* Farkas, E.; Hajdi, G.; Moro, A.; Hollos, I.; Palfi, A.; Wohlmuth, G. (Laszlo Munic. Hosp. Infect. Dis., Budapest, Hungary). *Infection* 2(4):196-200, 1974.

Infants with viral hepatitis were studied to determine the probability of diaplacental transmission of viral hepatitis on the basis of antigenemia. The 16 infants studied were 2-5 months old, 7 of whom were found to be positive for the Australia antigen (HB_sAg). A common feature of the cases was antigenemia of the mother and child. Routine tests were made to determine serum bilirubin level, SGOT and SGPT enzyme activity, also to determine HB_sAg, the related antibody and the circulating antigen-antibody complex. Diaplacental infection was evident in a case of hepatitis in a newborn baby delivered of an HB_sAg-positive mother having serum hepatitis during gestation or at delivery, or becoming HB_sAg-positive during her illness. It is concluded that maternal antigenemia is a potential hazard, and suggested that the definition of connatal viral hepatitis be revised on the basis of the identification of HB_sAg.

2226 HEPATITIS B IN AN ONCOLOGY UNIT. *(E.)* Wands, J. R.; Walker, J. A.; Davis, T. T.; Waterbury, L. A.; Owens, A. H.; Carpenter, C. C. J. (Johns Hopkins Univ. Sch. Med., Baltimore, Md.). *N. Engl. J. Med.* 291(26):1371-1375, 1974.

An epidemic of hepatitis B among the 91 personnel in an oncology ward is described. Acute icteric hepatitis (as shown by marked elevation of the glutamic oxalacetic transaminase and bilirubin values) developed in 9 of the 91 workers, and asymptomatic anicteric hepatitis in 1. In 19 additional personnel, there was evidence for asymptomatic exposure to hepatitis B surface antigen (HB_sAg) by a 4-fold or greater rise in pre-existing hepatitis B surface antibody (anti-HB_s) titer (as determined by hemagglutination inhibition and hemagglutination), and by seroconversion from anti-HB_s negative to positive. A 19.5% frequency of HB_sAg was detected in 87 tumor patients before, during, and after the epidemic. In 5 asymptomatic patients with pre-existing HB_sAg, unusually high titers (mean value of 1:128,000) developed on high doses of antitumor chemotherapy. One of these patients may have been a common source of exposure for 8 of the personnel. Because asymptomatic and anicteric HB_sAg-positive tumor patients represent a great risk to oncology-unit personnel, prophylactic measures and precautions should be instituted in these wards.

2227 SEVERE VIRAL HEPATITIS TYPE B IN INFANCY. *(E.)* Dupuy, J. M.; Frommel, D.; Alagille, D. (INSERM, Child. Hosp., Bicetre, France). *Lancet* I(7900):191-194, 1975.

Case histories are presented for 14 infants, aged 2-5 months, with severe acute viral hepatitis. The hepatitis B surface antigen (HB_sAg) and/or hepatitis B antibody (HB Ab), as determined by counter-immunoelectrophoresis, microcomplement fixation, inhibition of passive hemagglutination, or radioimmunoassay were present in 11 cases. Eight infants had received blood derivatives in the neonatal period, and HB_sAg-positive hepatitis developed in 7 of these, suggesting a causal relationship between the hepatitis and the carrier state of the mother. In all patients, the prothrombin time was 10% or less of normal and the levels of SGPT and bilirubin were increased. Liver biopsies showed widespread necrosis without inflammatory cells. Despite intensive supportive therapy, exchange transfusions, and administration of anti-HB_sAg γ-globulins, the mortality rate was high (57%). Liver regeneration was shown by an increase in clotting factor V and the appearance of α-fetoprotein in the serum. Differences in incubation time found in some cases suggested an oral route of infection in those with the longer incubation period, rather than a parenteral route. The study showed that infants can completely eliminate the hepatitis B virus after showing all the signs of hepatitis type B. The outcome of the disease seemed to be influenced by the route of infection, dose of the virus, presence of maternal antibodies, and possibly genetic susceptibility.

2228 OBSERVATIONS ON ACUTE PHASE AND FOLLOW-UP OF SIMILAR SERIES OF CASES OF HB-Ag POSITIVE AND HB-Ag NEGATIVE HEPATITIS. *(E.)* Chiaramonte, M.; Dardanoni, L.; Farini, R.; Filippazzo, G.; Genova, G.; Naccarato, R.; Pagliaro, L.; Spano, C. (Dep. Med., Univ. Palermo, Italy). *Rend. Gastroenterol.* 6(1):1-5, 1974.

A comparison between 177 patients with HB Ag-positive (HB Ag+) acute hepatitis and 169 patients with HB Ag-negative (HB Ag-) acute hepatitis was carried out. The cases were followed until complete recovery, or death, or until a pathologically defined type of chronic liver disease developed. A preliminary analysis showed a similar distribution of the patients of the HB Ag^+ and HB Ag- groups for age and sex and for initial values of SGOT, SGPT, and bilirubin. Diagnosis of chronic aggressive hepatitis or cirrhosis fulfilled the generally accepted criteria and was ultimately confirmed or excluded by histological examination. The sera of all patients with a chronic sequelae were examined by direct immunofluorescence for anti-nucleus, anti-smooth muscle, and anti-mitochondria antibodies, and by the latex test for anti-IgG antibodies (rheumatoid factor). HB Ag in sera was detected by immunodiffusion, high voltage counter electrophoresis and quantitative complement fixation. Both the acute initial attack and the chronic sequelae of hepatitis appeared more severe in the HB Ag+ cases. In the group of 177 HG Ag+ patients, 5 died during the acute jaundice phase and 4 developed cirrhosis, whereas none in the group of 169 HB Ag- patients died or became cirrhotic. The 12 patients with HB Ag+ acute hepatitis who developed chronic aggressive hepatitis were predominantly males (10 of 12) and 7 of 12 received steroids during the acute jaundice phase. Results of this study support earlier conclusions that, after an acute attack of HB Ag+ hepatitis, the persistence of HB Ag in blood seems associated with continuing liver disease, without any instance of chronic post-jaundice healthy carrier state. It is suggested that the healthy carrier state may be connected with an impairment of the mechanism causing clearance of HB Ag from the blood stream. All patients who developed chronic sequelae following an acute attack of HB Ag+ hepatitis remained chronically HB Ag+, suggesting that in these patients the persistence of HB Ag in the organism plays a determinant role. These data also confirm the usefulness of the HB Ag assay as a prognostic tool.

2229 VIRAL HEPATITIS AND ITS MULTIPHASIC COURSE: STUDY OF 704 CASES FOLLOWED FOR 6-12 MONTHS. *(Fr.)* Laverdant, C.; Cristau, P.; Essioux, H.; Molinie, C.; Malvy, J. L.; Alleaume, D. (Begin Mil. Hosp., Saint-Mande, France). *Arch. Fr. Mal. App. Dig.* 63(4):305-314, 1974.

Clinical symptoms and serum bilirubin, alkaline phosphatase, γ-globulin, the thymol turbidity test, SGPT, and hepatitis B antigen (HB Ag) were followed for 6 to 12 months in 704 patients (682 men and 22 women, mean age 23 yr) out of 778 hospitalized with viral hepatitis. Patients with fulminating hepatitis, cholestatic hepatitis, and hepatitis with an atypical onset were excluded. HB Ag was present in 15.2% of all patients and was not related to the course of the disease in these patients. According to the clinical symptoms and liver function tests, the course of viral hepatitis was monophasic in 67% of the patients, i.e. after reaching a maximum, clinical and biochemical disorders became progressively less pronounced until recovery was complete. In the remaining 33% of the patients, the course was multiphasic. Patients with a multiphasic course were classified into 3 subgroups: (1) 42.70% with one rebound occurring during the 3rd week and reaching a maximum around the 35th day; (2) 41.15% with one rebound occurring during the 4th week and reaching a maximum around the 50th day; and (3) 16.15% with 2 rebounds, the second of which occurred between the 50th and the 80th day. In contrast to relapses, which are defined as the recurrence of a new, complete set of clinical, biological, and pathological symptoms, identical to those observed at the onset, rebounds are only temporary biological disorders accompanied by nonspecific clinical symptoms (asthenia, anorexia, failure to regain lost weight). Relapses were less common than rebounds, occurring in only 8 of the original 778 patients. Among patients with a multiphasic course, a correlation was found between SGPT values and biopsy findings. Rebounds were accompanied by connective tissue lesions, particularly inflammatory infiltrates in the portal triad; little or no hepatocyte necrosis was observed. All of the patients with multiphasic courses recovered completely so that no specific treatment is justified.

2230 ACUTE FULMINANT HEPATITIS: A CLUSTERING OF CASES. *(E.)* Sheinbaum, A. J.; Damus, K. H.; Michael, T. D.; Gitnick, G. L. (Univ. California Los Angeles Cent. Health Sci.). *Arch. Intern. Med.* 134(6):1093-1094, 1974.

During a 10-wk period in a rural county, 48 cases of viral hepatitis were reported. Out of these 48 cases, 10 cases developed fulminant hepatitis. Seven of the fulminant hepatitis cases were studied to determine the contribution of host factors, environment, viral mutation or epidemiologic conditions to its occurrence. Hepatitis B antigen (HB Ag) subtyping by rheophoresis was carried out on the 5 patients who were HB Ag-positive; 4 of these were subtyped *ay* and 1 was *ad*. All patients were relatively young (18-26 yr old) and 4 were known parenteral drug users. There was no common water supply and, except for the possibility that 2 patients may have shared needles, there was no evidence that any of the other people were exposed to each other or to a common source. The 20.8% incidence of acute fulminant hepatitis among the 48 reported cases of acute viral hepatitis represents an extremely high morbidity in a relatively short time period (presently accepted rate is only 2%). This high incidence may represent a "clustering" of cases which is not a unique phenomenon but actually represents a common occurrence that was previously undetected. Two explanations are suggested for this clustering: 1) a unique but unidentified combination of host and environmental factors; and 2) a spontaneous viral mutation resulting in the development of an unusually virulent hepatitis virus strain whose virulence was unrelated to the presence or absence of HB Ag or any 1 of its subtypes.

2231 AUSTRALIA ANTIGEN IN HOSPITAL PATIENTS. *(Ger.)* Zollikofer, J. (Limmattal Blood Donor Cent., Schlieren/Zurich, Switzerland). *Z. Gastroenterol.* 12(3):201-208, 1974.

2232 TREATMENT OF SEVERE AND PROLONGED FORMS OF TYPES A AND B VIRAL HEPATITIS. *(Fr.)* Benhamou, J. P. (Beaujon Hosp., Clichy, France). *Cah. Med.* 14(9):805-810, 1973.

2233 CLINICAL EXPERIENCE WITH POLILEVO (PILLS) IN LIVER THERAPY. *(Ger.)* Rizzo, F.; Arcuri, F. (Munic. Hosp., Genoa, Italy). *Therapiewoche* 24(17):1957-1958, 1960, 1974.

2234 CELL-MEDIATED IMMUNITY TO HEPATITIS B ANTIGEN IN BLOOD DONORS WITH PERSISTENT ANTIGENAEMIA OR HIGH TITRE ANTIBODY. *(E.)* Lee, W. M.; Reed, W. D.; Mitchell, C. G.; Eddleston, A. L. W. F.; Dymock, I.; Williams, R. (King's Coll. Hosp., London, England). *Gut* 15(10):825, 1974.

2235 LIVER FUNCTION AND BLOOD CLOTTING IN PATIENTS WITH VIRAL HEPATITIS. *(Rus.)* Fedenko, Iu. I. (Voroshilovgrad Med. Inst., USSR). *Klin. Med. (Mosk.)* 52(8):102-107, 1974.

2236 LAPAROSCOPIC ASPECTS OF SUBACUTE NECROTIZING HEPATITIS. *(Ger.)* Knoblauch, M.; Lammli, J. (Dep. Intern. Med., Univ. Zurich, Switzerland). *Z. Gastroenterol.* 12(1):50, 1974.

2237 SOME HEMODYNAMIC PARAMETERS IN VIRAL HEPATITIS IN CHILDREN. *(Rus.)* Chernova, I. V.; Dik, G. A. (Chelyabinsk Med. Inst., USSR). *Vopr. Okhr. Materin. Det.* 18(12):13-15, 1973.

2238 BLOOD CLOTTING PARAMETERS IN EVALUATING THE SEVERITY OF HEPATOCYTE INSUFFICIENCY IN VIRAL HEPATITIS. *(Rus.)* Zhitar, V. D. (Kishinev Med. Inst., USSR). *Klin. Med. (Mosk.)* 52(11):93-97, 1974.

See also, 1919, 1920, 2178.

LIVER AND BILIARY TRACT

Chronic Hepatitis

2239 HEPATITIS B ANTIGEN AND ANTIBODY IN ACTIVE CHRONIC HEPATITIS AND OTHER LIVER DISEASES IN AUSTRALIA. A MULTICENTER COLLABORATIVE STUDY. *(E.)* Cooksley, W. G. E.; Powell, L. W.; Mistilis, S. P.; Mackay, I. R.; Barker, L. F. (R. Brisbane Hosp., Australia). *Am. J. Dig. Dis.* 20(2):110-114, 1975.

Radioimmunoassay for hepatitis B antigen (HB Ag) and anti-HB Ag was used to study the possibility that persistent antigen is involved in the pathogenesis of chronic active hepatitis. Sera from 85 patients with active chronic hepatitis, 83 with other liver diseases, 98 with non-liver diseases, and 67 healthy controls were assayed. HB Ag and anti-HB Ag were each found in 3 of the 85 active chronic hepatitis patients (4%). Of the other 248 patients, 9 were HB Ag-positive; 7 of these had liver disease other than chronic active hepatitis (8%). Seventeen of the 248, including 5 with liver disease other than chronic active hepatitis (6%), 8 from the non-liver disease patients (8%) and 4 from the controls (6%), were anti-HB Ag-positive. It is concluded that the low prevalence of the HB Ag indicates persistent hepatitis B virus infection is rarely relevant to pathogenesis of active chronic hepatitis in Australia.

2240 CHRONIC PERSISTENT HEPATITIS: PATHOLOGICAL AND CLINICAL STUDY OF 100 PATIENTS. *(Fr.)* Sidi, S.; Michel, H.; Peters, R.L.; Redeker, A. G. (John Wesley Hosp., Los Angeles, Calif.). *Arch. Fr. Mal. App. Dig.* 63(4):285-295, 1974.

Clinical, biochemical, serological and, in 89 cases, histological studies were performed systematically over a 2-12-yr period on 100 patients, 16-80 yr old (mean age 31 yr), who developed chronic persistent hepatitis as a sequela of acute icteric or anicteric hepatitis. All patients had transaminase levels of more than 50 Karmen U for at least 1 yr after the initial episode of hepatitis; none were alcoholics or had other chronic liver diseases. Few of these patients had signs of hepatic insufficiency; 16% had hepatomegaly and 3%, splenomegaly, but none had associated portal hypertension. Although SGOT and SGPT activities were increased, serum bilirubin, protein, and prothrombin levels were within normal limits in all or almost all cases; 30 min BSP retention was abnormal in 18% but was not related to transaminase activity. No relation was found between transaminase levels and severity of histological lesions. Punch liver biopsies showed minor histological lesions: swollen hepatocytes with a cobblestone appearance; apparent obliteration of the sinusoidal lumen; little or no fibrosis and necrosis; and dilation of the portal spaces with round cell infiltrates. These lesions were suggestive of a viral etiology which was confirmed in 30% by the presence of hepatitis B antigen; hepatitis B antibodies were detected in 5 patients, but they persisted in only 1. No patients had lupus erythematosis cells. Long-term followups revealed that none of these patients developed chronic aggressive hepatitis or cirrhosis. Transaminase levels remained the same in 32%, increased in 12%, and decreased in 56%. Although transaminase levels returned to normal after 3-10 yr in 30% of these patients, histological lesions never regressed completely. A controlled study of 40 patients demonstrated that prednisone (30 mg/day for 2 months) had no effect on serum transaminase levels which were likewise not affected by rest or exercise.

2241 THE REACTION OF SERUM ENZYMES AND METABOLITES TO A STANDARDIZED WALKING EXERCISE IN 140 PATIENTS WITH CHRONIC LIVER DISEASE. *(Ger.)* Muting, D.; Hammes, P. H.; Ecknauer, R. (Prof. Kalk Spec. Clin. Liver Dis., Bad Kissingen-Hausen, Germany). *Munch. Med. Wochenschr.* 116(43):1873-1880, 1974.

Serum enzymes and metabolites were measured in 20 normal subjects and 140 patients with chronic liver diseases 1 and 24 hr after the subjects performed a standardized walking test (6 km in 60 min). The enzymes measured were sorbitol dehydrogenase (SDH), glutamic dehydrogenase (GLDH), SGOT, SGPT, and creatine phosphokinase (CPK), and the metabolites were glucose, lactate, triglycerides, plasma ammonia, and free serum phenols. All enzyme and metabolite levels increased in the controls, and SDH, GLDH, and ammonia levels increased in patients recovering from hepatitis. Changes in SDH, GLDH, and SGPT following physical exercise were in the same direction in almost all patients. The GLDH activity increased in patients with chronic persistent, and decreased in those with chronic aggressive hepatitis. Increased plasma ammonia levels and inversion of the SGOT:SGPT ratio were observed in patients with complete cirrhosis and little inflammation. Patients with chronic aggressive hepatitis had the most pronounced increases in the activities of all enzymes and in ammonia levels. In patients with fatty liver, GLDH was the most sensitive indication of hepatocyte damage. Lactate levels were highest in obese patients with fatty liver who engaged in little physical activity. Except for patients with chronic aggressive hepatitis and pronounced inflammation, walking exercise was tolerated well by all patients. Improvements in the results of these tests occurred during recovery from hepatitis.

2242 CELL-MEDIATED IMMUNITY TO HUMAN TAMM-HORSFALL GLYCOPROTEIN IN AUTOIMMUNE LIVER DISEASE WITH RENAL TUBULAR ACIDOSIS. *(E.)* Tsantoulas, D. C.; McFarlane, I. G.; Portmann, B.; Eddleston, A. L. W. F.; Williams, R. (King's Coll. Hosp. Med. Sch., London, England). *Br. Med. J.* 4(5943):491-494, 1974.

Cell-mediated immune responses to Tamm-Horsfall glycoprotein isolated from human urine were investigated using the leukocyte migration test. The 71 patients studied included 24 with active chronic hepatitis, 24 with primary biliary cirrhosis, and 23 with various liver diseases not attributable to autoimmunity. These final 23 patients included those with alcoholic cirrhosis (6), liver damage from paracetamol overdose (4), idiopathic hemachromatosis (3), halothane hepatitis (2), primary hepatocellular carcinoma (2), extrahepatic obstructive jaundice (2), carbon tetrachloride liver injury (1), liver damage from *Amantia phalloides* (2), and hepatic hydatid disease (1). Sixteen healthy controls were also studied. None of the patients had overt renal tubular acidosis (RTA) but 7 of 24 patients with active chronic hepatitis (29 (29%) and 4 of 24 with primary biliary cirrhosis (17%) had the incomplete type. Abnormal leukocyte migration responses were found in 91% of the patients with active chronic hepatitis or primary biliary cirrhosis with an associated renal tubular acidosis (inhibition in 5 and stimulation in 5) but in only 19% of those without renal tubular acidosis. Among the 23 patients with non-autoimmune liver diseases, there were only 2 who had abnormal migration indices; in the control group, the results were within normal limits. In addition, using an immunofluorescent technique with rabbit antibody to Tamm-Horsfall glycoprotein, the presence of a material reacting immunologically as Tamm-Horsfall was shown in the human liver cell membrane. These findings suggest that the development of an immune response to this glycoprotein, initiated by release of cross-reacting antigens from damaged hepatocytes, could be the mechanism underlying the occurrence of renal tubular acidosis in some patients with autoimmune liver disease.

2243 CHRONIC AGGRESSIVE (LUPOID) HEPATITIS: DIAGNOSIS, DIFFERENTIAL DIAGNOSIS, AND THERAPY. *(Ger.)* Berg, P. A.; Brandt, H. (Med. Clin., Univ. Tubingen, Germany). *Therapiewoche* 24(6):521-522, 525-526, 528, 531-532, 534, 537-538, 540, 543-544, 1974.

2244 ELECTROCARDIOGRAPHY IN PATIENTS WITH CHRONIC HEPATITIS AND CIRRHOSIS. *(Rus.)* Olbinskaia, L. I.; Gorbatenko, V. P.; Migachev, V. N. (I. M. Suchenov 1st Moscow Med. Inst., USSR). *Klin. Med. (Mosk.)* 52(8):113-115, 1974.

See also, 2168, 2186.

LIVER AND BILIARY TRACT

Alcoholic Liver Disease

2245 TWO NEW TESTS FOR THE EARLY DETECTION OF ALCOHOLIC CIRRHOSIS. *(Fr.)* Belanger, M.; St. Hilaire, D.; Lessard, A. (Chicoutimi Hosp., Canada). *Union Med. Can.* 103(9):1557-1563, 1974.

Comparisons of blood protein fractions were made on (1) 43 patients with clinical and biological signs of alcoholic cirrhosis (mean age 54 yr), (2) 400 chronic male alcoholics, aged 18-74 yr (mean age 49 yr) with no clinical signs of active cirrhosis, and (3) 200 controls, aged 15-60 yr with no clinically diagnosed disease which could affect the results.

All but 6 of the cirrhotics had IgA levels of 400 mg/100 ml or higher (mean 642 mg/100 mg) which were accompanied, in some cases, by increases in IgG levels. Transferrin concentrations (mean 146 mg/100 ml) were low in all of the cirrhotics, averaging 53% of normal, and a correlation was found between transferrin and albumin levels. The ratio of IgA:transferrin ranged from 2 to 16 in this group. IgA and transferrin levels and the IgA:transferrin ratio provided an accurate indication of the extent of fibrosis. Large variations were found in ceruloplasmin, haptoglobin, and antitrypsin concentrations, and the test for α_1-fetoprotein was negative on all of these cirrhotics. Of the 400 chronic alcoholics, 337 had no significant protein abnormalities associated with alcohol abuse while 63 had IgA levels of 394 mg/100 ml or higher and decreased transferrin concentrations; the ratio of IgA:transferrin averaged 3.41 with an upper limit of 7.18 in the latter group. Ceruloplasmin concentrations were low, but this might have been caused by factors other than alcoholism. It is recommended that IgA and transferrin concentrations and the IgA:transferrin ratio be used to diagnose alcoholic cirrhosis in its early stages.

2246 ANALYSIS OF THE RESULTS FROM CLEARANCE OF CONTINUOUSLY PERFUSED INDOCYANINE GREEN IN PRECIRRHOTIC AND CIRRHOTIC ALCOHOLICS. *(Fr.)* Traissac, F. J.; Paccalin, J.; Montantin, C. (St. Andre Hosp., Bordeaux, France). *J. Med. Lyon* 54(1241): 101-108, 1973.

A "pre-cirrhotic" state of alcoholics was characterized according to the hepatic involvement as shown by the clearance curve of indocyanine green. The subjects included 13 control subjects, 7 declared alcoholics who presented no clinical symptoms, 7 alcoholics with nondecompensated cirrhosis, and 19 alcoholics with decompensated cirrhosis. Indocyanine green was perfused at 3 different concentrations and arterial blood samples began to be drawn after 30 min. The optical density of the plasma was measured, and the plasma volume was determined by radioionized albumin. The differences between the constant of proportionality between plasma concentration and intrahepatic dye fixation for the alcoholic subjects as compared with the control subjects was not significant, but the calculated maximum amount of dye transported did drop significantly for the non-clinical alcoholics compared with the control subjects, and more for the clinically alcoholic subjects. The technique is still experimental, but the results suggest the indocyanine green test may be able to predict the prospects of liver cirrhosis by the lowering of the maximum amount of dye transported before the standard tests for liver function. The test has the advantage for research of sensitivity.

2247 PRESENCE OF SERUM α-1-FETOPROTEIN IN ALCOHOLIC HEPATITIS. *(E.)* Miller, A. I.; Moral, M. D.; Schiff, E. R. (VA Hosp., Miami, Fl.). *Gastroenterology* 68(2):381-383, 1975.

A case is presented of α_1-fetoprotein detected in association with alcoholic hepatitis. A 53-yr-old man with a history of chronic alcoholism presented with anorexia, malaise, and jaundice. Laboratory findings were consistent with a diagnosis of alcoholic hepatitis, but counterimmunoelectrophoresis of serum revealed the presence of α_1-fetoprotein to a dilution of 1:8. Liver scan, laparoscopy, and biopsy failed to disclose any malignancy. Treatment with bed rest and diuretics resulted in improved condition, and α_1-fetoprotein positive reaction disappeared from the serum, with no recurrence. It is suggested that in patients positive for α_1-fetoprotein, but displaying no other indications of neoplastic invasion, alcoholic hepatitis should be considered as a possible cause.

2248 INTRACTABLE ASCITES IN ALCOHOLIC CIRRHOSIS: TREATMENT BY REINJECTION OF ASCITES FLUID COMBINED WITH DIURETICS. *(Fr.)* Perrin, D.; Bredoux, P.; Arzul, C.; Bergerault, P.; Dedieu, P.; Miniconi, P. (St. Jacques Hosp., Nantes, France). *Arch. Fr. Mal. App. Dig.* 63(5):365-373, 1974.

Intractable ascites accompanying alcoholic cirrhosis in 32 patients (23 men and 9 women) was treated by aspiration of ascites fluid which was then infused i.v. at rates ranging from 300 to 1200 ml/hr (mean 645 ml/hr). The total fluid infused ranged from 1.5 to 8 liters/day (mean 5.77 liters/day). This technique was used in 37 hospitalizations of these 32 patients and was combined with bed rest, a low-sodium diet (22 mEq/day), and KCl (1-3 g/day) in all cases; 16 patients also received prednisolone (20-30 mg/day). Infusions of ascites fluid were given without diuretics in 50 cases and with p.o. or i.v. diuretics in 136. Furosemide (mean dose 60 mg/day) and spironolactone (mean dose 350 mg/day) were given, either alone or in combination. Half of the patients received antibiotics (ampicillin or penicillin, colistin or gentamycin) systemically on the days of infusion. Short-term followups showed that treatment was ineffective in 4 cases; all of these patients died with kidney failure, uremia, severe hyponatremia, and hyperkalemia. Some improvement occurred in 10 patients, but 2 died of gastrointestinal bleeding and 3 of kidney failure; only 1 of 3 patients who developed recurrent ascites several weeks after treatment benefitted from repeated infusions of ascites fluid. Ascites disappeared for long periods in 18 patients (23 hospitalizations), but 2 of these patients died of hemoperitoneum and gastrointestinal bleeding, resp. Long-term followups of 18 patients revealed that 15 are alive and in good health 1 yr after treatment. Long-lasting results were obtained only when infusion of ascites fluid was combined with administration of diuretics, particularly spironolactone, and a low-sodium diet. It is recommended that 6 liters/day of ascites fluid be infused at a rate of 600 ml/hr and that spironolactone (100-400 mg) be given 3 or 4 days/week. Antibiotics did not appear to prevent infections; it is suggested that corticosteroids be discontinued.

2249 STUDY OF CHRONIC FIBRINOLYSIS IN ALCOHOLIC CIRRHOSIS. *(Fr.)* Thuot, C.; Perron, L. (St. Luc Hosp., Montreal, Canada). *Union Med. Can.* 103(8):1400-1404, 1974.

The effect of heparin (200 U/kg/24 hr infused i.v. for at least 48 hr) on blood clotting and fibrinolysis was studied in 12 patients with alcoholic cirrhosis complicated by chronic fibrinolysis. Patients were hospitalized for hepatic decompensation which was associated, in some cases, with alcoholic hepatitis. None of these patients had secondary obstructive jaundice and none had bleeding with the exception of ecchymosis or rare nosebleeds. Heparin rapidly corrected fibrinolysis in 6 of these patients and had no effect in the remaining 6 cases. Patients who responded to heparin therapy initially had moderate thrombopenia; moderately reduced fibrinogen; chronic fibrinolysis, as demonstrated by accelerated plasma euglobulin lysis; a slight increase in fibrinogen degradation products; a prolonged thrombin time; and moderately decreased plasminogen. Factors V and VIII were within normal limits, and no evidence of paracoagulation (cryofibrinogen and soluble complexes) was found. Heparin corrected the rate of fibrinolysis and increased fibrinogen levels rapidly and increased the platelet count slowly until it reached the lower normal range in 4 patients. However, in some patients accelerated fibrinolysis recurred 48 hr after discontinuation of heparin infusion. The findings suggest that patients responding to therapy had intravascular coagulation with secondary physiological lysis. The failure of heparin to correct fibrinolysis in the remaining 6 patients suggests that they had systemic or primary fibrinolysis with no evidence of disseminated intravascular coagulation.

2250 CLINICAL PROBLEMS OF ALCOHOLIC CIRRHOSIS. I. BIOLOGICAL AND CLINICAL FEATURES OF ALCOHOLIC CIRRHOSIS. *(Ser.)* Knezevic, S.; Falisevac, V.; Kallai, L.; Hadzic, N.; Pichler, E.; Spicer, F. (Med. Fac., Zagreb, Yugoslavia). *Acta Med. Iugosl.* 28(4):339-353, 1974.

2251 ALCOHOLIC FATTY LIVER. *(It.)* Pasquazzi, M. (St. Filippo Neri Hosp., Rome, Italy). *Policlinico* [*Prat.*] 81(1):1-13, 1974.

2252 ALCOHOLIC CARDIOMYOPATHY WITH HYPERFERREMIA CAUSED BY EXCESSIVE GASTROINTESTINAL ABSORPTION OR IRON. *(Fr.)* Auzepy, P.; Valette, H.; Albessard, F.; Deparis, M. (Bicetre Hosp., Paris, France). *Ann. Med. Interne* 125(12):923-926, 1974.

See also, 2253.

LIVER AND BILIARY TRACT

Cirrhosis

2253 HETEROZYGOUS ALPHA-ANTITRYPSIN DEFICIENCY AND CIRRHOSIS IN ADULTS, A FORTUITOUS ASSOCIATION. *(E.)* Morin, T.; Feldmann, G.; Benhamou, J. P.; Martin, J. P.; Rueff, B.; Ropartz, C. (Beaujon Hosp., Clichy, France). *Lancet* 1(7901):250-251, 1975.

The frequency of the different genotypes of serum α_1-antitrypsin deficiency was compared in 169 cirrhotic and 394 healthy subjects. Cirrhosis was histologically proven in 105 and was of apparent alcoholic origin in 132. There was no significant difference between normal and cirrhotic patients in the frequency of protease inhibitor genotypes MM, MS, MZ or other minor ones, or the alleles M, S, Z, and the other alleles. It is concluded that previously observed correlations between cirrhosis and heterozygous α_1-antitrypsin deficiency are merely fortuitous.

2254 CIRRHOSIS ASSOCIATED WITH PARTIAL DEFICIENCY OF ALPHA-1-ANTITRYPSIN: A CLINICAL AND AUTOPSY STUDY. *(E.)* Craig, J. M.; Dunn, A. F. G.; Peters, R. L. (Univ. South. Calif. Sch. Med., Los Angeles). *Hum. Pathol.* 6(1):113-120, 1975.

A case history of a 63 yr old Mexican female with cirrhosis associated with a partial deficiency of α_1-antitrypsin is presented. The patient was admitted for treatment of uncontrolled diabetes and had a questionable history of esophageal varices in 1966. In 1971, the patient had hepatosplenomegaly with ascites, palmar erythema and a few spider angiomas. She denied ethanol abuse. Blood chemistry studies showed a serum albumin of 3.2 g%, serum globulin of 3.7 g% (α_1, 0.1 g%, α_2, 0.15 g%, β, 0.9 g%, γ, 2.2 g%), α_1-antitrypsin of 135 mg% (normal = 200-400 mg%), serum trypsin inhibitory capacity of 0.598 U (normal = 0.85-1.60), and normal serum bilirubin, SGOT and SGPT levels. P_I typing showed an SZ phenotype. Tests for hepatitis B antigen, L.E. cells, mitochondrial, antinuclear and smooth muscle antibodies and α-fetoprotein were negative. Liver biopsy revealed eosinophilic cytoplasmic inclusions in a few hepatocytes and features suggestive of cirrhosis. The patient was later readmitted in hepatic coma with gastrointestinal bleeding and died. At autopsy, the liver contained multiple nodules. The eosinophilic cytoplasmic inclusions were again noted in formalin-fixed sections and were strongly positive to diastase digested PAS and with alcian blue. Fluorescence microscopic studies with labeled antibody to α_1-antitrypsin showed positive vesicular structures within the cytoplasm of hepatocytes at the periphery of the nodules with decreasing fluorescence in hepatocytes closer to the center of the nodules. All hepatocytes contained

large amounts of dilated granular endoplasmic reticulum cisternae filled with low density homogeneous flocculant material. These findings suggest that at least 1 of the heterozygous P_i phenotype α_1-antitrypsin deficiencies may be associated with hepatic cirrhosis in adults.

2255 OXYGEN TRANSPORT IN NON-HYPOXAEMIC CIRRHOTIC PATIENTS. *(E.)* Verdier, F.; Hakim, J.; Sraer, J.; Fag, M.; Boucherot, J.; Troube, H.; Drutel, P. (INSERM, Beaujon Hosp., Paris, France). *Biomedecine* 21(6):268-271, 1974.

Oxygen transport was studied in 11 cirrhotic patients without hypoxia, but who were hypocapnic and anemic. In addition to arterial O_2 and CO_2 partial pressures, O_2 saturation and pH, intraerythrocyte ATP, 2,3-diphosphoglycerate and O_2 partial pressure at 50% saturation were also determined. Results of arterial blood gases showed a mean O_2 partial pressure of 93.1 Torr and a mean O_2 saturation of 97.9%. Patients, however, were alkalotic (plasmatic pH of 7.47; intraerythrocyte pH of 7.25), hypocapnic (CO_2 partial pressure Torr) and anemic (hemoglobin of 10.6 gm%). Blood ATP levels were decreased from the control value of 4.33 µM/gm hemoglobin to 3.05 µM/gm. 2,3-Diphosphoglycerate levels were increased to 19.2 µM/gm hemoglobin (control = 14.0 µM/gm.) Partial O_2 pressure at 50% saturation was also significantly increased to 28.9 Torr (control = 25.9). There was no significant correlation between 2,3-diphosphoglycerate levels and partial O_2 pressure at 50% saturation. These patients thus exhibited a lowered O_2 affinity, as previously reported, which ensures a good O_2 supply to the tissues. The alkalosis was apparently responsible for the increased 2,3-diphosphoglycerate levels observed.

2256 EFFECT OF ORNITHINE α-KETOGLUTARATE ON HIGH BLOOD LEVELS OF AMMONIA INDUCED IN CIRRHOTICS. *(Fr.)* Peter, B.; Imler, M.; Tongio, J.; Schlienger, J. L.; Stahl, J. (Univ. Hosp. Cent., Strasbourg, France). *Ann. Gastroenterol. Hepatol.* 10(3):179-196, 1974.

Continuous determination of blood ammonia levels were performed on 20 fasting male cirrhotics, aged 35-80 yr, using a Technicon Autoanalyzer before and after ingestion of 5 g ammonium acetate dissolved in 100 ml water. Of these patients, 19 had clinically compensated cirrhosis and 7 had decompensated cirrhosis (jaundice and/or ascites); only 1 had a transitory hepatic precoma following gastrointestinal bleeding several weeks before. Blood ammonia levels returned to their initial values within 2 hr in all these subjects. When ammonia levels returned to their initial values, 16 cirrhotics were given a second dose of ammonium acetate followed immediately by i.v. infusion of 25 mg ornithine α-ketoglutarate (OAKG), dissolved in 250 ml physiological saline, over a 1 hr period; 10 cirrhotics served as controls. Within 1-5 min after ingestion of ammonium acetate, arterial ammonium concentrations increased, and this increase persisted for 60-120 min. Curves differed greatly from one patient to another with ammonia levels reaching a maximum 5-93 min after ingestion of ammonium acetate. Peripheral venous levels of ammonia were always lower than arterial levels, and the difference between arterial and venous levels showed great individual variation. Infusion of OAKG in 16 cirrhotics after ingestion of ammonium acetate resulted in a variable and incomplete decrease in arterial ammonia levels which averaged on the order of 34%. Simultaneous recordings of arterial and venous ammonia levels in 9 cirrhotics failed to demonstrate that OAKG had any effect on ammonia utilization in the muscles. In 3 other cases, simultaneous recording of ammonia levels in arterial and subhepatic venous blood showed that OAKG stimulates hepatic utilization of ammonia but has no effect on portal blood shunted around the liver by portacaval anastomoses. From a practical standpoint, the therapeutic effect of OAKG on high blood ammonia levels in cirrhotics is limited by liver function and, even more, by perihepatic shunting of portal blood. For this reason, the use of OAKG is not recommended for reducing high blood ammonia levels found in hepatic coma.

2257 BILE ACID METABOLISM IN CIRRHOSIS. IV. CHARACTERIZATION OF THE ABNORMALITY IN DEOXYCHOLIC ACID METABOLISM. *(E.)* Yoshida, T.; McCormick, III, W. C.; Swell, L.; Vlahcevic, Z. R. (VA Hosp., Richmond, Va.). *Gastroenterology* 68(2): 335-341, 1975.

Cirrhotic and non-cirrhotic patients were studied to determine the mechanism of faulty deoxycholic acid metabolism in cirrhosis. Eight cirrhotic, and 5 normal males were given 5 Ci of ^{14}C-cholic or deoxycholic acid by enema, and 4 cirrhotic males were given 450 mg/day of cholic acid p.o. for 3 days. In addition, fecal bile acids were determined in 12 controls and 9 cirrhotic patients not given exogenous bile acids. In the cirrhotic patients given the deoxycholic acid enemas the bile level of that acid showed a significant increase after 2-4 hr, while the chenodeoxycholic acid level was not affected. Normal controls showed no effect. After 24 hr, 95% of the deoxycholic acid still retained the label in all cases, but the appearance of the label in the bile was much slower in the cirrhotic patients. In cirrhotic patients given the cholic acid enemas, bile composition was not affected, though some label was found in the bile. It was also noted that in the most severe cases of cirrhosis, cholic acid was converted to deoxycholic acid much more slowly. Of the 4 given cholic acid p.o., deoxycholic acid levels were raised slightly in 2 patients with originally low levels, and unchanged in those with normal original levels (2). Biliary cholic acid levels were increased in all 4. In the fecal sample experiments, it was found that cirrhotics had a significantly lower % deoxycholic acid level than did controls, while the secondary bile acid levels were significantly higher. Lithocholic acid levels were the same in both groups. The results suggest that impaired intestinal absorption may account for biliary deoxycholic deficiency in cirrhosis, and lack of cholic acid substrate is probably not a major factor. Fecal analysis suggests that some cirrhotic patients may be deficient in colonic

bacteria or enzymes that favor conversion of cholic to deoxycholic acid.

2258 PROGNOSTIC VALUE OF THE IgA:TRANSFERRIN RATIO IN CIRRHOSIS. *(Fr.)* Paris, J.; Jaillard, J.; Paris, J. C.; Sezille, G.; Fruchart, J. C.; Voiment, Y. M. (no affil.). *Bull. Acad. Natl. Med. (Paris)* 158(1):96-99, 1974.

Radial immunodiffusion was used to measure IgA and transferrin, and differences in the IgA:transferrin ratio were analyzed in 132 adults including: (1) 13 with acute hepatitis; (2) 11 with chronic cirrhogenous hepatitis; (3) 13 with primary or secondary liver cancer; (4) 80 with alcoholic cirrhosis; and (5) 15 controls with no evidence of liver disease or chronic alcoholism. Patients with alcoholic cirrhosis were further classified into 4 subgroups: (a) 14 chronic alcoholics with some clinical and biological signs suggestive of cirrhosis, but no jaundice, edema, ascites, or gastrointestinal bleeding; (b) 30 patients with known cirrhosis who responded to diuretics; (c) 19 cirrhotics who had at least 2 episodes of decompensation but who were in satisfactory condition; and (d) 17 with rapidly evolving cirrhosis who died in hepatic coma within several months. No patients who died of gastrointestinal bleeding were included in group (d). Comparison of mean IgA:transferrin values showed no significant difference between patients with acute hepatitis (1.60) and controls (1.9). Highly significant increases were found in chronic cirrhogenic hepatitis (3.92) and liver cancer 2.48). In alcoholic cirrhosis, IgA:transferrin values ranged from 3.64 in group (a) to 15.10 in group (d). Decompensation of cirrhosis was accompanied by a significant increase in the mean value. Determination of the IgA:transferrin ratio is recommended as a method for establishing both a diagnosis and prognosis for alcoholic cirrhosis.

2259 USE OF BUMETANIDE IN THE TREATMENT OF ASCITES DUE TO LIVER DISEASE. *(E.)* Moult, P. J. A.; Lunzer, M. R.; Trash, D. B.; Sherlock, S. (R. Free Hosp., London, England). *Gut* 15(12):988-992, 1974.

Seventeen patients with ascites from chronic liver disease were treated with bumetanide to evaluate its performance and side effects. All had daily sodium and potassium intakes of 22 and 50 mEq, resp., a daily fluid intake of 1 liter. Body weight was stabilized and the drug initially administered at a dose of 1 mg/day and varied later (in the range of 1/2-4 mg/day) according to weight loss, urine output and sodium excretion. Patients were examined daily for hepatic encephalopathy, weighed, and had urine specimens collected for 24 hr volume estimation and electrolyte composition. Plasma electrolytes, urea and postprandial blood sugar were estimated 3 times/week. Thirteen responded to the initial 1 mg dose with a weight loss of 0.58 kg/day, 1 patient responded to a dose of 2 mg, and 1 died of hemorrhaging esophageal varices. In 2 others, diuresis occurred, but sodium excretion was less than intake. One responded to a 2 mg dose, but the other died of bleeding from esophageal varices. Over an average of 21 days of treatment, weight loss averaged 6.4 kg/day. No significant changes were found in blood glucose, uric acid, creatinine, calcium, phosphate, bilirubin, aspartate transaminase, or alkaline phosphatase. Six patients required a potassium-sparing diuretic to achieve control. It is concluded that bumetanide is an effective treatment for ascites in liver disease, but all patients should recieve supplementary potassium chloride or potassium-sparing diuretics.

2260 DETERMINATION OF THE PATENCY OF A SPLENORENAL SURGICAL ANASTOMOSIS. CREATININE AND UREA AS INERT MARKERS OF THE PORTAL BLOOD STREAM. *(E.)* Gips, C. H.; Que, G. S.; Trip, J. A. J. (Univ. Hosp., Groningen, Netherlands). *Acta Hepatogastroenterol. (Stuttg.)* 21(5):346-351, 1974.

Shunt patency was assessed in 4 patients with cryptogenic macronodular cirrhosis following splenorenal anastomosis for repeated bleeding from esophageal varices. Patency was determined from measurements of simultaneous arterial and left renal venous blood following loading with 5 g ammonium acetate p.o. In 2 patients in whom both pre- and postoperative studies were conducted, the elevation of arterial ammonia was smaller before (100-200 µg%) than after (>400 µg%) surgery, suggested shunt patency. Catheterization of these 2 patients postoperatively revealed hepatic sinusoidal pressures (wedged hepatic venous free pressure) of 5 and 12 mm Hg (normal, 0-3). Following p.o. loading with creatinine (5 g) and urea (20-30 g) in 1 patient, the resp. concentrations in the left renal vein and artery showed the same patterns of increase as did the ammonia level after loading, whereas hepatic vein concentrations were lower. This patient was subsequently found to have a patent shunt on autopsy following death from hepatocellular carcinoma. These studies suggest that ammonia, urea and creatinine loading may be used to assess shunt patency as well as direction of main portal blood flow following splenorenal anastamosis: if renal vein creatinine of urea concentration after loading exceeds hepatic vein concentration, blood flow is hepatofugal; if *vice versa*, flow is hepatopetal.

2261 COMPARATIVE STUDY OF BASAL ARTERIAL AMMONEMIA AND OF ORALLY INDUCED HYPERAMMONEMIA IN CHRONIC PORTAL SYSTEMIC ENCEPHALOPATHY, TREATED WITH NEOMYCIN, LACTULOSE, AND AN ASSOCIATION OF NEOMYCIN AND LACTULOSE. *(E.)* Pirotte, J.; Guffens, J. M.; Devos, J. (Baviere Hosp., Liege, Belgium). *Digestion* 10(6):435-444, 1974.

The effects of neomycin and lactulose, separately and in combination, were studied with respect to ammonia metabolism in chronic portal systemic encephalopathy in 9 cirrhotic patients. The encephalopathy was corrected and the patients stabilized with regard to urea level, blood pH, kalemia, and hepatic biology, by means of a salt-free, low protein diet. Once stabilized, the subjects were given 15 g lactulose 3 times/day for 5

days followed by 5 days of no treatment. When the effects of lactulose had worn off, they were given 250 mg neomycin 4 times/day for 10 days, then lactulose (45 g) was added to the neomycin for the next 12 days. Fasting arterial ammonia, and oral ammonia load tests were performed regularly during the different periods of treatment. Both the neomycin and the lactulose individually were found to reduce significantly the basal ammonemia to the same degree. The combination of both, however, reduced the values significantly lower than either by itself. The oral load tests showed that neomycin improved ammonia tolerance slightly, while lactulose improved it significantly, and both together had a significantly greater effect than either alone. It is concluded that both drugs together reduce colonic absorption more than either alone, presumably because the neomycin in short term small doses spares the colonic flora that break down lactulose into its active ingredients.

2262 THE HEMODYNAMICS OF PORTAL HYPERTENSION REVISITED: DETERMINANTS AND SIGNIFICANCE OF OCCLUDED PORTAL PRESSURES. *(E.)* Moreno, A. H.; Burchell, A. R.; Reddy, R. V.; Panke, W. F.; Nealon, Jr., T. F. (St. Vincent's Hosp., New York, N.Y.). *Surgery* 77(2):167-179, 1975.

Portal blood flow was measured by square wave electromagnetic flowmeter in 153 cirrhosis patients about to be submitted to portacaval anastamosis. In addition, occluded portal pressures were determined by manometry in a subgroup of 80 patients and portal and hepatic artery flows were measured in 50 patients. Portal vein flow averaged 447 ml Hg/min and free portal pressure, 28.5 mm Hg (387 mm H_2O), in agreement with earlier findings. The incidence of stagnant flows (only to and from motions synchronous with respiration) was only 8.5%. Hepatic and splanchnic pressures measured after occlusion of the portal vein averaged 320 and 441 mm H_2O, resp. In 9 of these patients, hepatic occluded portal pressure was higher than either or both the free portal pressure and the splanchnic occluded pressure; however, 7 had forward flow into the liver from 80-1116 ml Hg/min and flow in the remaining 2 was stagnant. Analysis of these results indicated that alteration of the hepatic occluded portal pressure with respect to splanchnic occluded portal pressure is primarily determined by hepatic arterial resistance and hepatic venular outflow resistance on one side and splanchnic arterial resistance on the other. It is concluded that spontaneous reversal of portal flow in cirrhosis does not necessarily occur when hepatic occluded portal pressure exceeds either or both the free portal pressure or the splanchnic occluded portal pressure as measured in this study.

2263 PRIMARY BILIARY CIRRHOSIS: DIFFICULTY IN ESTABLISHING AN EARLY DIAGNOSIS. *(Fr.)* Hofstetter, J. R.; Realini, S.; Fontolliet, C.: Gardiol, D. (Canton. Hosp., Lausanne, Switzerland). *Schweiz. Med. Wochenschr.* 104(50):1880-1882, 1974.

Of 10 women with primary biliary cirrhosis, 2 were diagnosed in the early stages. At onset both patients had pruritus, antimitochondrial antibodies, and increased serum alkaline phosphatase levels; 1 had increased IgM levels. Biopsies showed portal infiltration, bile duct lesions and, in 1 case, granulomas with giant cells; no signs of cholestasis were evident. Administration of azathioprine caused the pruritus to disappear and laboratory findings to improve in both patients, but when therapy was discontinued, exacerbations developed. Therefore, immunosuppressive therapy should probably be continued over a long period. Primary biliary cirrhosis can be differentiated from chronic hepatitis by increases in IgM and alkaline phosphatase, the absence of cytolysis, and the presence of antimitochondrial antibodies. It is important to diagnose primary biliary cirrhosis in its early stages so that immunosuppressive therapy can be instituted.

2264 LIVER SCHISTOSOMIASIS AND PRIMARY CIRRHOSIS. A CLINICO-PATHOLOGICAL STUDY. *(E.)* Berthoud, S.; Cox, J. N.; Thiebaud, G.; Bouzakoura, C. (Dep. Med., Univ. Geneva, Switzerland). *Virchows Arch. [Pathol. Anat.]* 364 (3):285-294, 1974. (3):285-296, 1974.

The case of a 54-yr-old Swiss housewife suffering concomitantly from liver schistosomiasis and primary biliary cirrhosis is presented. The patient had traveled to Morocco, Madeira and the Canary Islands during recent holidays. A needle liver biopsy revealed, among other lesions, the presence of calcified fragments of schistosome ova in a portal tract. Laboratory findings revealed a raised erythrocyte sedimentation rate of 54/90 mm, hemoglobin of 14.5 g%, WBC of 5950/mm^3, and platelet count of 198000/mm^3. Urinalysis was normal. There were no bile pigments. Liver function tests were perturbed, showing in particular an increased alkaline phosphatase and the presence of antimitochondrial antibodies. The possibility of an autoimmune type of hepatic affection triggered by the presence of the schistosome eggs is suggested. Since the date of onset of either conditon, bilharzia or primary biliary cirrhosis, cannot be determined, it is suggested for the future that autoantibodies be determined, particularly antimitochondrial antibodies, not only in primary biliary cirrhosis and other chronic liver diseases, but also in cases of schistosomiasis of the liver.

2265 CIRRHOSIS: ITS MORPHOLOGICAL AND CLINICAL-CHEMICAL FEATURES. *(Ger.)* Vido, I.; Schmidt, E.; Schmidt, F. W. (Med. Coll., Hannover, Germany). *Z. Gastroenterol.* 12(1):51, 1974.

2266 THE EFFECT OF PORTOCAVAL SHUNT ON COLON TRANSFER OF AMMONIA IN CIRRHOSIS OF THE LIVER. *(E.)* Sein, M. M.; Hardy-Smith, A.; Shields, R. (Dep. Surg., Univ. Liverpool, England). *Gut* 15 (10):837, 1974.

2267 "PSEUDOSURGICAL" ACUTE ABDOMEN IN CIRRHOSIS. *(Fr.)* Pedreira, J. D.; Villalonga, C.; Martinez-Vazquez, J. M.; Guardia, J.; Tornos, J.; Bacardi, R. (no affil.). *Cah. Med.* 15(3):131-135,1974.

2268 THE LYMPHATIC SYSTEM AND LYMPHATIC CYSTS IN THE CAPSULE OF THE CIRRHOTIC LIVER. *(Ger.)* Mielke, F.; Wegener, H. (Westend Clin., Free Univ., Berlin, Germany). *Z. Gastroenterol.* 12(3):169-178, 1974.

2269 RETROGRADE SPLENOPORTOGRAPHY. *(E.)* Wannagat, L. (Metab. Clin., LVA, Wurttemberg, Germany). *Acta Hepatogastroenterol. (Stuttg.)* 21(4):278-289, 1974.

2270 DIAGNOSIS OF ACUTE BLEEDING FROM ESOPHAGEAL OR GASTRIC VARICES IN PATIENTS WITH PORTAL HYPERTENSION. *(Rus.)* Shertsinger, A. G.; Tsorikaev, V. O. (All-Union Sci. Res. Inst. Clin. Exp. Surg., Moscow, USSR). *Klin. Khir.* (11):30-33, 1974.

2271 CURRENT DIAGNOSIS AND PREOPERATIVE THERAPY FOR PORTAL HYPERTENSION WITH BLEEDING AND NONBLEEDING ESOPHAGEAL VARICES. *(Ger.)* Schulz, D.; Kaufer, C.; Frommhold, H.; Paquet, K. J. (Surg. Clin., Rhenish Friedrich Wilhelm Univ., Bonn, Germany). *Therapiewoche* 24(5):385-386, 1974.

2272 HEMODYNAMIC CHARACTERISTICS OF DIFFERENT FORMS OF PORTAL HYPERTENSION (FROM ANGIOGRAPHIC DATA). *(Rus.)* Kuzin, M. I.; Savchenko, A. P.; Filimonov, G. P.; Pomelova, L. A. (I. M. Sechenov 1st Moscow Med. Inst., USSR). *Klin. Med. (Mosk.)* 51(11):20-26, 1973.

2273 WHICH PATIENTS WITH PORTAL HYPERTENSION SHOULD UNDERGO SHUNT SURGERY? *(Fr.)* Forster, E. (no affil.). *J. Med. Strasbourg* 4(11): 741-744, 1973.

2274 EXPERIENCE IN THE SURGICAL TREATMENT OF PATIENTS WITH PORTAL HYPERTENSION. *(Rus.)* Zubov, D. S. (M. Gorky Med. Inst., Donetsk, USSR). *Klin. Khir.* (9):35-40, 1974.

2275 EMERGENCY TREATMENT OF HEPATIC COMA AND PORTACAVAL ENCEPHALOPATHY. *(Fr.)* Imler, M. (Univ. Hosp. Cent., Strasbourg, France). *Bull. Soc. Sci. Med. Grand Duche Luxemb.* 110(2):239-255, 1973.

2276 DIALECTICS IN THE CURING OF ASCITES IN CIRRHOSIS BY INTEGRATING TRADITIONAL CHINESE MEDICINE WITH WESTERN MEDICINE. *(Ch.)* Lin, T. K. (no affil.). *Kexue Tongbao* 19(8):349-351, 1974.

2277 VIRAL AND BACTERIAL ANTIBODIES IN PRIMARY BILIARY CIRRHOSIS AND OTHER CHRONIC LIVER DISEASE. *(E.)* Thomas, H. C.; Holden, R.; Ironside, J.; Somervilld, R. (Dep. Bacteriol. Immunol., Univ. Glasgow, Scotland). *Gut* 15(10):826, 1974.

2278 HYPERCATABOLISM OF THIRD COMPONENT OF COMPLEMENT IN PRIMARY BILIARY CIRRHOSIS. *(E.)* Potter, B. J.; Elias, E.; Jones, E. A. (R. Free Hosp., London, England). *Gut* 15(10):837, 1974.

2279 LAPAROSCOPIC FINDINGS IN PRIMARY BILIARY CIRRHOSIS. *(Ger.)* Schmid, M. (Waid Munic. Hosp., Zurich, Switzerland). *Z. Gastroenterol.* 12(1):48, 1974.

See also, 1941, 1942, 2168, 2172, 2186, 2196, 2241, 2242, 2244, 2249, 2250.

LIVER AND BILIARY TRACT

Gallbladder and Biliary Tract Diseases

2280 INTERPRETATIVE PROBLEMS IN ENDOSCOPIC RETROGRADE CHOLANGIOPANCREATOGRAPHY. *(E.)* Block, M. A.; Schuman, B. M.; Weckstein, M. L. (Henry Ford Hosp., Detroit, Mich.). *Am. J. Surg.* 129(1): 29-33, 1975.

Of 41 patients with biliary tract problems studied by endoscopic retrograde cholangiopancreatography, interpretation of ductograms or endoscopic observations could not be accomplished with ease in 10. Although incomplete filling of the bile ducts usually signifies organic obstruction, complete filling was not achieved in 1 patient with the subsequent diagnosis of intrahepatic cholestasis and another with a known liver hamartoma and subsequent diagnosis of concomitant hepatitis. When obstructing lesions were present, their specific origin and histological character could not be delineated. A pancreatic carcinoma produced only partial obstruction of the bile duct sufficient to cause jaundice in 1 patient; in a patient with jaundice associated with pancreatitis, an endoscopic pancreatogram was consistent with this diagnosis, but a choledochogram could not be obtained. Other limitations of endoscopic cholangiography included difficulties in demonstrating minute stones in 2 patients, in recognizing anatomic variations that caused the pancreatic duct to be confused with the bile duct in 1 case, and in defining the status of recurrent strictures in a patient with Roux-en-Y loops. On endoscopic examination, inflammatory changes in the duodenal papilla simulated carcinoma in 1 case while an ampullary carcinoma simulated an inflammatory process in another. In a 3rd patient, changes at the ampulla produced by carcinoma at the

head of the pancreas were interpreted as a probable ampullary carcinoma. It is noted that pancreatic ductograms were obtained in 2 patients with carcinoma of the ampulla of Vater and dilation of the pancreatic duct was observed in 2 patients with stones in the common bile duct. Biopsy of ampullary carcinomas did not provide histologic confirmation in 2 patients. When interpretative difficulties occur in endoscopic retrograde cholangiopancreatography, the findings should be correlated with other available clinical information.

2281 CARCINOMA OF THE GALLBLADDER. A STUDY OF 181 CASES. *(E.)* Ohlsson, E. G.; Aronsen, K. F. (Malmo Gen. Hosp., Sweden). *Acta Chir. Scand.* 140(6):475-480, 1974.

The incidence of carcinoma of the gallbladder was analyzed during a 15-yr period in a well-defined and non-selected population served by 1 hospital and the clinical course of the disease followed. During the 15-yr period, 181 cases were diagnosed, an incidence of about 5/100,000 inhabitants/yr. There were 148 female and 33 male, 80% of whom were above 60 yr old. Gallstones were present in 88.4% of the patients. The carcinoma was very difficult to diagnose in an early phase. In 19% of the patients operated upon, the indication for surgery was cholelithiasis. In 53% the indication for surgery was jaundice, and in 18% it was a clinically suspect malignant disease in patients without jaundice. At the time of operation 20% of the cases were not diagnosed as carcinoma by the surgeon but by the pathologist who later examined the gallbladder. Of all patients undergoing operation, 34.5% were cholecystectomized. The rest were subjected to explorative laparotomy or to some type of surgical palliative procedure. The mean survival time of those cholecystectomized was 15.8 months and for those subjected to surgical exploration only 1.5 months. Only 3 patients survived for a longer time and all of these had carcinoma *in situ*. It is concluded that only carcinoma *in situ* can be treated radically and that in order to obtain better therapeutic results, carcinoma of the gallbladder must be diagnosed earlier than it presently is.

2282 A CLINICOPATHOLOGICAL REVIEW OF 68 CARCINOMAS OF THE GALLBLADDER. *(E.)* Donaldson, L. A.; Busuttil, A. (Dep. Pathol., Univ. Glasgow, Scotland). *Br. J. Surg.* 62(1):26-32, 1975.

Sixty-eight gallbladder carcinomas were reviewed with regard to diagnosis and treatment. Fifty-one had symptoms and signs of acute or chronic cholecystitis, of which 25 had a palpable right hypochondrial mass, and 18 had jaundice. Long standing biliary disease was found in 33 patients by radiological and surgical methods, and the disease exceeded 1 yr in 25. No substantial weight loss was seen, even in extremely advanced cases. In 10 patients, the serum alkaline phosphatase test was positive, though there was no sign of biliary obstruction. It is suggested that partial hepatic resection be considered in treatment. It is concluded that long standing biliary disease and high serum alkaline phosphatase levels are diagnostic indications of gallbladder carcinoma.

2283 DOUBLE TRANSHEPATIC DRAINAGE. *(Fr.)* Praderi, R.; Delgado, G.; Mazza, M.; Gomez Fossati, C.; Davidenko, N. (Manuel Quintela Hosp., Montevideo, Uruguay). *Lyon Chir.* 70(5):294-298, 1974.

Two transhepatic tubes were used to anastomose the right and left hepatic ducts separately to the intestine in 9 patients. These patients consisted of 3 with cancers involving the junction of the hepatic ducts and 3 with postoperative stenosis of the hepatic ducts which, in 2 cases, was recurrent. This technique was also used in 3 patients with recurrences of inoperable cancer. The use of these tubes simplified suturing of the right and left hepatic ducts to the intestine: 4 or 5 sutures were usually enough. This technique made it possible to control the caliber of the right and left hepaticojejunal anstomoses separately and prevented sutures from coming out by reducing pressures in the biliary tract and anastomosed intestinal loop. By combining this technique with transhepatic drainage through a double U-tube, it is possible to change and clean the tubes if they disintegrate or become clogged.

2284 INTRAHEPATIC CHOLANGIOENTERIC ANASTOMOSIS IN CARCINOMA OF THE HILUS OF THE LIVER. *(E.)* Bismuth, H.; Corlette, M. B. (Paul Brousse Hosp., Villejuif, France). *Surg. Gynecol. Obstet.* 140(2): 170-178, 1975.

The validity of 45 intrahepatic cholangioenteric anastomoses for carcinoma of the hilus of the liver was tested regarding complications and long term results. Cholangioenteric anastomosis is defined as mucosa to mucosa approximation of a bile duct to the intestine. Of the 45 patients, the anastomosis was to the left in 40, to the right in 3 and bilateral in 2. On the left, all were to the duct of the left anterolateral segment and 28 were by the round ligament approach. In the first postoperative month there were 2 deaths, 1 from renal failure and 1 from cholangitis. Of the remaining patients the average survival time was 13 months. Of 15 who died under 6 months, metastases were known to be present during surgery in 12. Long term complications were recurrence of jaundice in 7 and biliary infection in 5. It was found that good cholangiography is essential for confirmation of diagnosis, and for choosing the site for the anastomosis. Recurrent jaundice in 4 cases was attributable in part to poor cholangiogram interpretation. It is felt that this surgical procedure using the left anteriolateral duct has given excellent results, especially with the round ligament technique, and is recommended in cases of this type.

2285 HEPATIC PORTOENTEROSTOMY AND ITS MODIFICATIONS FOR 'NONCORRECTABLE' BILIARY ATRESIA. *(E.)* Kasai, M. (Tohoku Univ. Sch. Med., Sendai, Japan). *Paediatrician* 3(3/5):204-212, 1974.

A follow-up study was conducted on survivors of biliary atresia of the "noncorrectable" type who had been treated by hepatic portoenterostomy. The operation was designed to remove the gallbladder and bile ducts replaced by fibrous tissue, or the tissue an-

terior to the portal vein when ducts were not identified, up to the porta hepatis, after which the bowel was anastomosed to the porta hepatis, using the duodenum or the jejunum in Roux-en-Y fashion. Of 57 patients on whom the operation was performed, 8 died during convalescence. Among the 49 survivors, active excretion of bile appeared after operation in 19 and was poor or nonexistent in the remaining 30 patients. Long-term survivors (10 of 19) were examined in March 1972, 1-17 yr after operation. Growth was within normal limits in 9 of 10. One patient showed marked retardation of physical growth and was diagnosed as having Turner's syndrome. Liver function tests showed nearly normal values except for 1 case of elevated bilirubin, 1 of elevated transaminase, and 1 of abnormal cephalin flocculation. Needle biopsy at varying intervals after operation indicated that changes of the hepatic parenchyma and ductular proliferation were improved rather rapidly, whereas recovery from fibrosis was slow and occasionally incomplete. Postoperative excretion of bile was not observed in any patient who had no remnant of the hepatic duct and was poor in those who had identifiable but fibrotic radicles. Patients having small patent radicles or fibrous mass at the porta hepatis, however, showed active excretion of bile after operation in about 50% of cases. There was also a close relationship between post-operative excretion of bile and size of bile ducts found on histological examination of the porta hepatis. Postoperative cholangitis was the primary cause of unfavorable results in patients who showed active excretion immediately after operation. When the surgical technique was modified to a double-Y hepatic portojejunostomy with external fistula, signs of cholangitis disappeared in 10 of 11 cases. Modifications of the hepatic portoenterostomy have now been carried out on 19 patients, 18 of whom survived. Postoperative bile excretion was good in 7 of 8 patients younger than 70 days but in only 2 of 10 older than 70 days. It is concluded that the majority of patients with biliary atresia may be curable if an adequate operation is carried out within the first 10 weeks of life.

2286 PREVALENCE OF CHOLELITHIASIS IN IDIOPATHIC LATE-ONSET IMMUNOGLOBULIN DEFICIENCY. *(E.)* Díaz-Buxo, J. A.; Hermans, P. E.; Elveback, L. R. (Mayo Clin. Found., Rochester, Minn.). *Ann. Intern. Med.* 82(2):213-214, 1975.

Fifty patients were studied to determine whether there was a correlation between idiopathic late-onset immunoglobulin levels below 30% of normal and an onset of cholelithiasis at least 7 yr after birth. Gallbladder roentgenograms or observations during surgery or autopsy were made on 37 (47%). Each of the 50 was classified according to the presence or absence of cholelithiasis at the last examination. A control group consisted of 500 patients who underwent abdominal surgery for benign upper gastrointestinal conditions. Twelve out of 50 (24%) with immunoglobulin deficiency had cholelithiasis, as opposed to 47 of the 500 controls (9.4%), a significant difference. It is concluded that there is an association between this type of immunoglobulin deficiency and cholelithiasis. It is suggested that the cholelithiasis developed because of a) a local abnormality in the gallbladder or b) decreased bile acid pool due to either excess intestinal loss of bile acids secondary to intestinal malabsorption or to a primary hepatic abnormality producing lithogenic bile.

2287 DISSOLUTION AND RETRIEVAL OF RETAINED COMMON DUCT STONES. *(E.)* Patterson, H. C.; Hothem, A.; Bream, C. (Dep. Surg., Univ. North Carolina, Chapel Hill). *Am. Surg.* 41(1):48-50, 1975.

Twenty-one cases of retained biliary duct stones were treated with 1 or a combination of 3 new techniques. First, dissolution was attempted with a continuous drip of 2500 U of lung heparin in 250 cc of saline, every 6 hr. Choledochography was done every 4 days. If the stones were unaffected, they were flushed with sodium cholate (30 mm/hr). Diarrhea was prevented during this treatment with cholestyramine. The cholate treatment was alternated with the heparin treatment every 24 hr when the patients showed intolerance to the straight cholate. To overcome inadequate bathing of the stones, rocking beds were used. If treatment was ineffective after 2 weeks, heparin dosage was doubled. If there was no improvement after 4 weeks, mechanical retrieval was attempted with a ureteral stone basket. This was effective on stones under 1 cm in size. Only 5 of the 21 cases were unsuccessful. It is concluded that the use of heparin, cholic acid, and the ureteral stone basket are effective means of dealing with retained stones.

2288 CHOLESTEROL KINETICS IN PATIENTS WITH CHOLESTEROL GALLSTONES BEFORE AND DURING CHENODEOXYCHOLIC ACID TREATMENT. *(E.)* Pedersen, L.; Arnfred, T.; Thaysen, E. H. (North Aalborg Hosp., Denmark). *Scand. J. Gastroenterol.* 9(8):787-791, 1974.

The cholesterol kinetics in 4 patients with radiolucent gallstones was studied by an isotope dilution technique before and after 3 months treatment with 500 mg chenodeoxycholic acid (CDC) daily. None of the patients had any history of obstructive biliary disease, cholecystitis, diabetes mellitus, or liver disease. A single dose of about 30 μCi of 1-α,2-α-^{3}H-cholesterol was given i.v. and fasting plasma samples obtained at first twice, later once, weekly for about 10 weeks to determine cholesterol specific activity. The results were plotted against time and the decay curve analyzed in terms of a 2-pool model. On this basis, the size and turnover of the 2 exchangeable pools was estimated. The input into the rapidly miscible pool (A), which is assumed to approximate total cholesterol turnover, was calculated from these figures. Thereafter CDC was given for 3 months and the determination of cholesterol kinetics was repeated. The means of the rapidly miscible pool A, the more slowly miscible pool B, and the input into pool A were diminished by 19, 8, and 40%, resp., during treatment. Plasma cholesterol showed a slight tendency to increase and plasma triglyceride to decrease during treatment. The treatment did not cause any discomfort. None of the patients experienced diarrhea or biliary colic. Transient elevation of SGPT occurred in one patient, but SGOT, alkaline phosphatases, bilirubin, prothrombin time, whole blood count, and serum electrophoresis remained normal in all of them. The results sug-

gest that CDC feeding in man suppresses cholesterol synthesis, and that it does not increase the total exchangeable cholesterol mass. It is suggested that CDC feeding affects cholesterol metabolism as follows. The increased intestinal concentration of bile acids inhibits intestinal cholesterol synthesis and increases the bile acid pool, which inhibits the transformation of cholesterol to bile acids.

2289 EFFECTS OF MEALS AND INTERRUPTION OF ENTEROHEPATIC CIRCULATION OF FLOW, LIPID COMPOSITION, AND CHOLESTEROL SATURATION OF BILE IN MAN AFTER CHOLECYSTECTOMY. *(E.)* Soloway, R. D.; Schoenfield, L. J. (Mayo Clin., Rochester, Minn.). *Am. J. Dig. Dis.* 20(2):99-109, 1975.

In 4 patients (3 women and 1 man) studied after cholecystectomy and common duct exploration for cholesterol gallstones, bile salt and phospholipid output and bile flow increased 46%, 35%, and 40%, resp., in response to breakfast. Cholesterol output did not increase significantly. Simultaneously, cholesterol concentration decreased (mean, 12%) while phospholipid and bile salt concentrations did not change. The degree of cholesterol saturation of bile decreased in each study, whether or not the original ratio indicated cholesterol saturation; in 3 of 5 studies, an initially supersaturated bile became unsaturated with cholesterol in response to breakfast. No significant changes were noted after lunch. During interruption of enterohepatic circulation, studies with radiolabeled bile acids indicated that the increased bile acid output in response to meals was secondary to increased recirculation of intestinal bile acids rather than to *de novo* bile acid synthesis. This mechanism may account for the adequate delivery of bile salts and the decreased degree of cholesterol saturation of the bile postprandially in cholecystectomized patients.

2290 EFFECTS OF LOW DOSE CHENODEOXYCHOLIC ACID FEEDING ON BILIARY LIPID METABOLISM. *(E.)* Adler, R. D.; Bennion, L. J.; Duane, W. C.; Grundy, S. M. (Phoenix Indian Med. Cent., Arizona). *Gastroenterology* 68(2):326-334, 1975.

The mechanism by which chenodeoxycholic acid reduces the relative bile cholesterol content was investigated and compared with the actions of cholic acid. Ten healthy subjects were given a diet containing 40% of its calories as lard. Five of the subjects were studied as inpatients for 3 months. The 1st month served as a control period and no bile acids were administered. During the 2nd month, some subjects were given a daily p.o. dose (500 mg) of either chenodeoxycholic acid or cholic acid, and during the 3rd, the 2 groups were crossed over. One subject stayed only 2 months and received only chenodeoxycholic acid, and the remaining 4 were treated as outpatients and also received only chenodeoxycholic acid. Two outpatients had dosages of 1500 and 250 mg/day, resp., and 1 inpatient was given 1000 mg cholic acid. Lithogenicity was determined from bile aspirated after an overnight fast and calculated as either molar % of cholesterol or lithogenic index. Biliary lipid secretion rates were determined by a marker dilution technique using ^{14}C-cholesterol. Bile acid pool size was measured by infusion of ^{14}C-cholic acid into the duodenum, and bile acid composition was determined by gas-liquid chromatography. It was found that lithogenicity was reduced in 9 subjects, the molar % of cholesterol was 24% lower after chenodeoxycholic feeding than control values, and the lithogenic indices showed a comparable decrease. Cholic acid had no significant effect. Secretion rates were 23% lower after chenodeoxycholic acid feeding than after cholic acid feeding or controls, and a slight decrease in bile acid pool size occurred after chenodeoxycholic acid feeding, although cholic acid caused a slight increase. Both acids produced significant increases in the proportion of the bile acid administered. It is concluded that bile lithogenicity is reduced even by low chenodeoxycholic acid doses, due to decreased cholesterol secretion, and without increased bile acid secretion or pool size.

2291 DIFFERENCES IN BILE ACID EXCRETION IN SUBJECTS WITH HYPERCHOLESTEROLAEMIA, HYPERTRIGLYCERIDAEMIA AND OVERWEIGHT. *(E.)* Nestel, P. J.; Hunter, J. D. (John Curtin Sch. Med. Res., Australian Natl. Univ., Canberra). *Aust. NZ J. Med.* 4(5):491-496, 1974.

Bile acid excretion in 8 subjects with familial hypercholesterolemia and in 6 subjects with hypertriglyceridemia were compared with normal and overweight controls, resp. Diet was standardized in the hypercholesterolemic subjects and their controls. Blood was drawn from all subjects after a 14 hr fast and the plasma cholesterol and triglyceride concentrations measured. Feces were collected for bile acid content determination for a period of 8 days after 4 to 6 weeks of observation. Chromium oxide was taken by the subjects for 10 days preceding and throughout the fecal collection period as a marker to measure fecal transit time. It was found that bile acid excretion was significantly less in the hypercholesterolemic group (2.99 mg/kg/day) than in their controls (3.88 mg/kg/day). In the hypertriglyceridemic subjects bile acid excretion was significantly higher than any of the other 3 groups (6.07 mg/kg/day), while their overweight controls had the same excretion rate as the normal controls. It is concluded that familial hypercholesterolemia may represent diminished catabolism of cholesterol to bile acids, while hypertriglyceridemia exhibits increased cholesterol catabolism and turnover, which is independent of excess body weight.

2292 USE OF THE GALLBLADDER NECK IN THE RECONSTRUCTION OF THE BIFURCATION OF THE MAIN HEPATIC DUCTS. *(E.)* Philippakis, M.; Apostolidis, N.; Androulakakis, P.; Doundoulakis, N.; Legakis, N. (Hippocration Gen. Hosp., Athens, Greece). *Am. Surg.* 41(2):103-105, 1975.

The gallbladder neck and the cystic duct were used in the reconstruction of the hepatic duct bifurcation in a 58 yr old man who presented with mild jaundice and intense pruritus. A tumor of the extrahepatic

biliary tree was suspected, and a small adenocarcinoma was discovered upon surgery at the junction of the 2 main hepatic ducts. After excision of the tumor the infundibulum and body of the gallbladder were resected, and the neck anastomosed to the stump of the right hepatic duct. The stumps of the left and common hepatic ducts were then anastomosed. After 13 months there were no complications. It is suggested that this operation avoids the sequelae of cholangioenterostomy, and is therefore recommended for operations in this area when the gallbladder and cystic duct are in healthy condition.

2293 JEJUNAL MUCOSAL GRAFT: A SUTURELESS TECHNIC FOR REPAIR OF HIGH BILE DUCT STRICTURES. *(E.)* Wexler, M. J.; Smith, R. (St. George's Hosp., London, England). *Am. J. Surg.* 129(2):204-211, 1975.

The jejunal mucosal graft, a sutureless technique to solve the problems of biliary stricture repair, is described and evaluated. The technique uses the transhepatic tube principle along with a mucosal graft. The graft is formed by removing a seromuscular patch near the end of a Roux-en-Y loop of jejunum, creating a mucosal diverticulum. The biliary tree is entered at the porta hepatis and anchored to the Roux-en-Y loop through the diverticulum. The tube is then pulled back, carrying along the sleeve of jejunal mucosa to direct contact with the intrahepatic duct epithelium. Sixty-one repairs of this type were done in 50 patients, all of whom were referred after previous biliary tract operations. Morbidity was extremely low, the major complication being infection. Most of the postoperative assessments were made after over 18 months. It was found that 85% of the operations had an ultimately successful result with an operative mortality of 6%.

2294 AN UNUSUAL CASE OF PORTAL SYSTEMIC ENCEPHALOPATHY. *(E.)* Chowdhury, A. R.; Finestone, A. J. (Temple Univ. Hosp., Philadelphia, Pa.). *Am. J. Dig. Dis.* 20(2):176-181, 1975.

A 60-yr-old woman developed portal systemic encephalopathy of unknown etiology after the construction of a jejunoileal bypass for obesity. She had had 2 previous abdominal operations for small intestinal obstruction and incisional hernia 25 and 15 yr previously, resp. There were no esophageal varices or splenomegaly. Increase in dietary protein from 20 to 80 g/day caused blood ammonia level to rise from 121 to 253 µg/100 ml. The oral ammonia tolerance test also showed low tolerance. Selective celiac and superior mesenteric arteriography, and venous phase roentgenography demonstrated occlusion of the superior mesenteric and splenic veins with marked filling of the collateral circulation. It is suggested that this unusual complication of intestinal bypass may have been a result of previous abdominal surgery.

2295 ANTIBIOTICS IN BILIARY DISEASE. *(E.)* Keighley, M. R. B.; Drysdale, R.; Burdon, D. W.; Alexander-Williams, J. (Gen. Hosp., Birmingham, England). *Gut* 15(10):824, 1974.

2296 THE EFFECT OF FLORANTYRONE (ZANCHOL) ON THE COMPOSITION OF T-TUBE BILE. *(E.)* Kirkpatrick, J. R.; Eastwood, M. A.; Mitchell, W. D. (West. Gen. Hosp., Univ. Edinburgh, Scotland). *Gut* 15(10):824-825, 1974.

2297 EFFECT OF WHEAT BRAN ON BILE SALT METABOLISM AND BILE COMPOSITION. *(E.)* Pomare, E. W.; Heaton, K. W.; Lowbeer, T. S.; White, C. (R. Infirm., Bristol, England). *Gut* 15(10):824-825, 1974.

2298 THE HEPATOBILIARY SYSTEM IN CHRONIC GASTRITIS AND PEPTIC ULCER IN CHILDREN. *(Uk.)* Bilousov, Iu. V. (Ukrainian Inst. Postgrad. Med., USSR). *Pediatr. Akush. Ginekol.* (5):15-17, 1974.

2299 TEN YEARS OF BILIARY SURGERY AT A REGIONAL HOSPITAL. *(Ger.)* Heim, U.; Lanz, R.; Nussbaumer, A.; Peter, M.; Ramser, O. (Kreuzspital, Chur, Switzerland). *Helv. Chir. Acta* 41(5/6):529-531, 1974.

2300 STUDY OF THE INNER SURFACE OF THE BILE DUCT. *(Fr.)* Juvara, I.; Vereanu, I.; Huch, A. (Dr. I. Cantacuzino Hosp., Bucharest, Rumania). *J. Chir. (Paris)* 106(6):599-608, 1973.

2301 EFFECTS OF CERULEIN ON MOTILITY OF THE EXTRAHEPATIC BILIARY TRACT. *(It.)* Gulisano, G.; Modica, G. (Inst. Clin. Gen. Med., Med. Ther., Univ. Catania, Italy). *Fegato* 19(3):597-606, 1973.

2302 THE PATHOPHYSIOLOGY OF TRANSDUODENAL SPHINCTEROTOMY. *(Ger.)* Rehner, M.; Soehendra, N.; Kiffmeyer, D.; Henning, H. (Surg. Clin., Univ. Hamburg, Germany). *Z. Gastroenterol.* 12(3):185-190, 1974.

2303 LONG-TERM RESULTS OF COMMON BILE DUCT SPHINCTEROTOMY. *(Pol.)* Ratynski, B.; Wyrzykowska, A.; Borkowski, M.; Weslolowski, J. (Clin. Vasc. Surg., CMKP, Warsaw, Poland). *Wiad. Lek.* 27(21):1879-1880, 1974.

2304 DECOMPRESSION CHOLEDOCHOSTOMY THROUGH THE STUMP OF THE CYSTIC DUCT. *(Rus.)* Pilovskii, D. L.; Zhitnikova, K. S.; Rida, F. (Volgograd Med. Inst., USSR). *Vestn. Khir.* 113(11):35-40, 1974.

2305 CRITICAL CONSIDERATION OF CHOLEDOCHODUODENOSTOMY. *(Ser.)* Bukurov, S.; Tasic, D. (Med. Fac., Univ. Belgrade, Yugoslavia). *Srp. Arh. Celok. Lek.* 102(3/4):81-88, 1974.

2306 TUMORS OF THE AMPULLA OF VATER. *(Sp.)* Torre Eleizegui, L.; Prieto Amigo, J.; Galvan Bofill, A. (no affil.). *An. Med. (Barc.)* 60(3):271-285, 1974.

2307 CHOLELITHIASIS: A METABOLIC CONSEQUENCE OF ILEOSTOMY. *(E.)* Mair, W. S. J.; Hill, G. L.; Goligher, J. C. (Gen. Infirm., Leeds, England). *Gut* 15(10):830, 1974.

2308 TYPE IV GAMMAGLOBULINOPATHY AND CHOLELITHIASIS. *(Sp.)* Nunez Carril, J.; Rodriguez Cuartero, A.; Mora Lara, R. J. (Fac. Med., Univ. Granada, Spain). *Rev. Esp. Enferm. Apar. Dig.* 44(6):597-604, 1974.

2309 EFFECTS OF CHOLELITHIASIS ON THE LIVER: 100 CASES STUDIED BY SURGICAL LIVER BIOPSIES. *(Sp.)* Lopez-Yarto, J.; Larranaga Barrera, E.; Candia Sierra, A.; Garcia Ocana, A. (Gran Hosp. Estado, Madrid, Spain). *Rev. Esp. Enferm. Apar. Dig.* 44(6):563-574, 1974.

2310 RADIOLOGICAL ERRORS IN THE DIAGNOSIS OF CHOLELITHIASIS. *(Rus.)* Kravchuk, A. N. (Odessa Med. Inst., USSR). *Khirurgiia (Mosk.)* (3):108-110, 1974.

2311 TREATMENT OF CHOLELITHIASIS. *(Fr.)* Caroli, J. (St. Antoine Hosp., Paris, France). *Cah. Med.* 14(9):799-804, 1973.

2312 CHOLEDOCHOLITHIASIS: STATISTICS ON 220 CASES. *(Fr.)* Devic, G.; Gelain, J. (no affil.). *Lyon Chir.* 70(6):378-383, 1974.

2313 RESULTS FROM SURGERY FOR CHOLEDOCHOLITHIASIS OVER AN EIGHT-YEAR PERIOD. *(Fr.)* Goinard, P.; Barbe, C.; Streichenberger, R.; Chavant, B.; Mathon, C. (no affil.). *Lyon Chir.* 70(6):368-377, 1974.

2314 ON 1,272 OPERATIONS FOR GALLSTONES IN THE COMMON BILE DUCT. I. OVERALL STATISTICAL FINDINGS. *(Fr.)* Mallet-Guy, P. (no affil.). *Lyon Chir.* 70(6):361-367, 1974.

2315 CHOLELITHIASIS AND HIATUS HERNIA: DIAGNOSTIC AND THERAPEUTIC PROBLEMS. *(Ger.)* Schmidt, H. D. (Surg. Clin., Univ. Mainz, Germany). *Therapiewoche* 24(18):2022-2026, 1974.

2316 OBSTRUCTIVE JAUNDICE CAUSED BY ECHINOCOCCOSIS OF THE BILIARY TRACT. *(Rus.)* Tevdorarazde, O. M.; Toidze, O. Sh. (Tbilisi Med. Inst., USSR). *Khirurgiia (Mosk.)* (3):115-116, 1974.

2317 CHOLESTYRAMINE THERAPY IN CHOLESTATIC LIVER DISEASE OF CHILDREN. *(E.)* Nelson, R.; Murphy, G. M.; Edkins, S.; Nutter, S.; Anderson, C. M. (Inst. Child Health, Univ. Birmingham, England). *Gut* 15(10):825, 1974.

2318 ACUTE CHOLECYSTITIS ASSOCIATED WITH TYPHOID FEVER: IS IT AGAIN OF CURRENT IMPORTANCE? *(Fr.)* Sava, G.; Thomas, M.; Jaeck, D.; Dalcher, G.; Seror, J. (no affil.). *J. Med. Strasbourg* 4(11):735-739, 1973.

2319 ACUTE CHOLECYSTITIS AS A POSTOPERATIVE COMPLICATION. *(Rus.)* Ulmanis, Ia. L. (Riga Med. Inst., USSR). *Vestn. Khir.* 113(9):126-127, 1974.

2320 WATER AND ELECTROLYTE BALANCE IN CALCULOUS CHOLECYSTITIS COMPLICATED BY OBSTRUCTIVE JAUNDICE IN ELDERLY PATIENTS BEFORE AND AFTER SURGERY. *(Rus.)* Ashrafov, A. A. (N. Narimanov Med. Inst. Azerbaidzhan, Baku, USSR). *Klin. Khir.* (8):27-29, 1974.

2321 CARDIALGIA IN CALCULOUS CHOLECYSTITIS. *(Rus.)* Panfilov, B. K. (Med. Fac., Patrice Lumumba Peoples Friendship Univ., Moscow, USSR). *Klin. Med. (Mosk.)* 52(7):60-66, 1974.

See also, 1781, 1889, 1890, 1909, 1921, 1922, 2152, 2165, 2182, 2279.

GENERAL

2322 ISOLATION OF TWO MINIGASTRINS FROM ZOLLINGER-ELLISON TUMOUR TISSUE. *(E.)* Gregory, R. A.; Tracy, H. J. (Physiol. Lab., Univ. Liverpool, England). *Gut* 15(9):683-685, 1974.

A pair of gastrin tridecapeptides ("minigastrins") isolated from Zollinger-Ellison tumor tissue obtained at postmortem examination about 18 hr after death were examined. A partially purified aqueous extract of the tumor tissue (50 ml) was filtered through a column of Sephadex G-50 superfine grade 5 cm x 50 cm run at 4 C in 0.40 M ammonium bicarbonate solution saturated with toluene. Fractions of 5 ml were collected every 10 min and the UV absorption at 280 nm read in 2 mm cells. The fractions corresponding to the smallest molecular weight were chromatographed on an aminoethyl cellulose column. Quantitative amino acid determinations of the major fractions showed that both peptides had the same amino acid composition, that is, leucine, glutamic acid, alanine,

tyrosine, glycine, tryptophan, methionine, aspartic acid, and phenylalanine. In 1 peptide, the tyrosine residue present was sulfated (type II); in the other (type I) it was not. The proportion of type I and II was approximately 2:1, similar to that for the gastrins and the heptadecapeptides isolated from the same source and from human antral mucosa. Both minigastrins were potent stimulants of gastric acid secretion when injected i.v. into conscious dogs provided with denervated pouches of the gastric fundus. Immunological evidence obtained in previous studies indicated that both minigastrins are present as circulating forms of the hormone gastrin in patients with the Zollinger-Ellison syndrome and in fed normal subjects. It is concluded that these peptides correspond to the fragment 5-17 of the heptadecapeptides isolated from the same source.

2323 AMINO TERMINAL GASTRIN FRAGMENT IN SERUM OF ZOLLINGER-ELLISON SYNDROME PATIENTS. *(E.)* Dockray, G. W.; Walsh, J. H. (VA Wadsworth Hosp. Cent., Los Angeles, Calif.). *Gastroenterology* 68(2):222-230, 1975.

The molecular species of gastrin in the serum of 6 patients with Zollinger-Ellison syndrome were separated by gel filtration and ion exhange chromatography, and concentrations were estimated by radioimmunoassay using 2 antisera with different specificities. In addition to the previously described forms of gastrin, a new component was identified which was present in high concentrations and which has properties similar to the 1:13 fragment of G-17. Because of its pattern of immunoreactivity, it is assumed that this new component has an N-terminal portion closely resembling that of G-17. The new component was present in gastrinoma tumor tissue. Its concentration in serum of gastrinoma patients increases markedly when secretin is injected. Since its component was identified in tumor extracts, and since its concentration rapidly increases in serum after secretin, it seems likely that this molecule is stored by the tumor together with G-17 and G-34. The fragment may be synthesized in its own right, or it may be produced from the breakdown of G-17 and G-34 within the synthesizing cell. The finding of this new component and the possibility that it may be the 1:13 fragment of G-17, indicates the desirability of a search for gastrin tetrapeptide.

2324 CALCIUM AND SECRETIN-STIMULATED GASTRIN RELEASE IN THE ZOLLINGER-ELLISON SYNDROME. *(E.)* Kolts, B. E.; Herbst, C. A.; McGuigan, J. E. (Univ. Florida Coll. Med., Gainesville). *Ann. Intern. Med.* 81(6):758-762, 1974.

Serum gastrin and calcium concentrations were measured and compared before and after calcium and secretin infusions in 3 patients (all women, aged 45-58 yr) with the Zollinger-Ellison syndrome and 3 control subjects. Each person was given a constant 3 hr i.v. infusion of calcium gluconate (5 mg Ca/kg/hr) in 0.9% sodium choloride. Secretin was given to 2 patients at a rate of 9 U/kg, i.v., for 1 hr. In the 3rd patient secretin was infused for 1 min at 1 U/kg, i.v. Calcium infusion induced marked increases in serum gastrin concentrations in the Zollinger-Ellison patients (increases 3100 to 19,000 pg/ml) and modest increases in the control subjects, mean increase of 29 pg/ml. In each patient receiving calcium infusion, there was significant positive correlation between serum gastrin and calcium levels. Secretin also increased serum gastrin levels, ranging from 800 to 11,000 pg/ml, with the most marked increases observed in the 2 patients receiving the larger dose of secretin. However, there was not a positive correlation between serum gastrin and calcium. These studies affirm secretin-induced paradoxical augmentation of serum gastrin in patients with Zollinger-Ellison syndrome. Failure to show positive correlation between serum gastrin and calcium with secretin administration, as is readily demonstrable with calcium infusion, provides no support for serum calcium increases as the mechanism by which serum gastrin is increased after secretin infusion in patients with Zollinger-Ellison syndrome.

2325 JEJUNAL MICROFLORA IN MALNOURISHED GAMBIAN CHILDREN. *(E.)* Heyworth, B. Brown, J. (Med. Res. Counc. Lab., Fajara, Gambia). *Arch. Dis. Child.* 50(1):27-33, 1975.

To investigate the presence of bacterial overgrowth, jejunal intubation was carried out in 25 cases of malnutrition (15 boys and 10 girls, aged 9-34 months). Growth of bacteria greater than 10^5 organisms/ml was found in 22 children, 17 of whom had a history of chronic diarrhea. The remaining 8 children either had no diarrhea or were having an acute attack lasting several days. In the children with chronic diarrhea, *Escherichia coli*, bacteroides, and enterococci occurred more frequently, while streptococci occurred more frequently in those with acute diarrhea. Bacilli, staphylococci, micrococci, *Klebsiella*, pseudomonads, and *Candida* often occurred in both groups, but in larger numbers in those with chronic diarrhea. These findings support the theory that children with malnutrition have considerable bacterial contamination of the jejunum, and that this may be of etiological significance for much of the diarrhea seen in malnourished children. It is suggested that this bacterial contamination may also be important in the pathogenesis of malnutrition. The presence of intestinal parasites in these malnourished children is also noted. Although the effectiveness of antibiotics has a limited time span, those children who had high counts of bacteroides improved dramatically when the correct antibiotic was given. This would be of some help in areas where protein-calorie malnutrition appears to be seasonal. It may be that removing the child to a more sanitary environment could constitute correct treatment, permitting natural clearance of organisms from the jejunum. It was noted that even those with chronic diarrhea who were not given broad-spectrum antibiotics underwent an improvement in the diarrhea and gained weight while in the hospital.

2326 IMMUNOLOGIC RESPONSES IN MALNOURISHED CHILDREN. *(E.)* Neumann, C. G.; Lawlor, Jr., G. J.; Stiehm, E. R.; Swendseid, M. E.; Newton, C.; Herbert, J.; Ammann, A. J.; Jacob, M. (Univ. California Sch. Med., Los Angeles). *Am. J. Clin. Nutr.* 28(2):89-104, 1975.

Humoral and cellular immune function was studied in 76 malnourished Ghanian children (aged 6 months - 6 yr) and 41 controls. Subjects with protein-calorie malnutrition were divided into groups of severely malnourished (34) and moderately malnourished (42) subjects based on anthropometry, overt signs of kwashiorkor or marasmus, and serum albumin. Serum immunoglobulins (IgG, M, A, D, and E), antibody response to keyhole limpet hemocyanin and polyvalent pneumococcal polysaccharide and cutaneous delayed hypersensitivity response to monilia, streptokinase-streptodornase and phytohemagglutinin were determined. Also, *in vitro* lymphocyte responses to several dilutions of phytohemagglutinin were determined in 27 severely malnourished children, 14 moderately malnourished children, and 10 controls. Increased immunoglobulin levels were found in all 3 groups with the levels somewhat higher in the malnourished groups. The marked elevation of IgE levels in the severely malnourished group may reflect intestinal parasitism or disordered T-cell function. Antibody responses were equal in all 3 groups. Cutaneous delayed hypersensitivity was decreased in both of the malnourished groups. The lymphocytes of 5 of the 27 severely malnourished children gave an abnormally reduced *in vitro* response to phytohemagglutinin. All controls and moderately malnourished children responded normally; however, the maximal level of response was higher in controls. The principle finding of the study was that the diminished cellular immune function in protein-calorie malnutrition was in proportion to the severity of the malnutrition, with little or no impairment of humoral immunity. The increased number of infections likely to occur in malnourished children may perpetuate a vicious cycle of further malnutrition not only through reduction of food intake but through the negative nitrogen balance produced by the catabolic effect of measles and other infections. The infections may in themselves depress cell-mediated immunity, thus allowing new infections to emerge or existing ones to become more severe. The use of simple tests or observations such as absent cutaneous delayed hypersensitivity, total lymphocyte counts under 1,000 cell/mm^3, decreased tonsillar size or absence of tonsils and marked reduction in anthropometric measurements for age should alert the clinician to the possible existence of a cellular immune defect with increased vulnerability to infection.

2327 GASTROINTESTINAL PROTEIN LOSS IN RHEUMATOID ARTHRITIS, STUDIED WITH ^{51}CR-CHROMIC CHLORIDE AND ^{125}I ALBUMIN. *(E.)* Plantin, L. O.; Strandberg, O. (King Gustaf V Res. Inst., Stockholm, Sweden). *Scand. J. Rheumatol.* 3(4):169-173, 1974.

Gastrointestinal excretion was studied during 7 days of ^{51}Cr-activity following the i.v. administration of ^{51}Cr-chromium chloride in 12 patients (10 women and 2 men aged 22-72 yr) with rheumatoid arthritis and 16 healthy controls (12 women and 4 men aged 20-51 yr). Most of the patients were simultaneously studied with respect to albumin pools and catabolism by ^{125}I-labeled albumin. In the control subjects, the mean serum albumin concentration was 5.1 g/100 ml and the mean intravascular pool was 2.46 g/kg. The mean fractional catabolism of iodinated albumin was 7.8% of the intravascular pool. In these subjects this represented an albumin catabolism of 0.19 g albumin/24 hr/kg. The mean extravascular/intravascular pool quotient was 1.6. The mean extravascular pool was 172 g. The mean ^{51}Cr excretion during 7 days was 0.7% of the dose. In the rheumatoid arthritis patients, the serum albumin concentration was subnormal as was the intravascular albumin pool. Thus an albumin deficit was well established. The mean fractional catabolic rate of ^{125}I-albumin, 10.0% of the intravascular pool, was not significantly elevated. A comparison of the mean quantitative catabolism between the arthritis and control groups showed no statistical difference. One of the patients had a secondary amyloidosis and combined mitral rheumatic heart disease in addition to rheumatoid arthritis. She had considerable renal protein losses, a relatively and absolutely elevated albumin catabolism, and a reduced intravascular albumin pool. The extravascular/intravascular quotient was significantly higher in the arthritis group than in controls. The mean fecal ^{51}Cr loss during the 7 days was significantly higher in the arthritis group, 1.7% of the dose. The low normal chromium excretion makes it possible to assess even small elevations of the gastrointestinal protein loss. The small gastrointestinal protein loss could be a symptom of early amyloid vascular lesion in patients with long-standing rheumatoid arthritis. I.v. injection of ^{57}Cr-chromium chloride is a comparatively convenient method for the diagnosis and quantitation of gastrointestinal protein loss.

2328 PATHOPHYSIOLOGY OF SHIGELLA DIARRHEA IN THE RHESUS MONKEY: INTESTINAL TRANSPORT, MORPHOLOGICAL, AND BACTERIOLOGICAL STUDIES. *(E.)* Rout, W. R.; Formal, S. B.; Giannella, R. A.; Dammin, G. J. (Univ. Kentucky Coll. Med., Lexington). *Gastroenterology* 68(2):270-278, 1975.

Jejunal, ileal, and colonic water and electrolyte transport was studied in *Shigella flexneri* 2a-infected monkeys. Infected animals were divided into 3 groups: dysentery alone, diarrhea alone, or diarrhea and dysentery together. In controls, net water, sodium, and chloride absorption was seen in the jejunum, ileum, and colon. All infected animals demonstrated diminished colonic absorption or net colonic secretion. In monkeys with dysentery alone, colonic dysfunction was the only transport defect observed. In contrast, animals with diarrhea either alone or combined with dysentery, exhibited net jejunal secretion, although ileal transport was little affected. A severe colitis with intramucosal shigellae was observed in all symptomatic animals. In the jejunum or ileum, however, morphological changes were minimal and bacterial invasion was not

seen. Therefore, unlike the "toxigenic diarrheas", shigellosis is both a small and large intestinal disease. Mucosal invasion of the colon is essential to the development of a morphological and transport defect. Dysentery results from a colonic transport defect, while diarrhea is secondary to jejunal secretion superimposed on the defect in colonic absorption.

2329 IMPORTANCE OF A NEW VIRUS IN ACUTE SPORADIC ENTERITIS IN CHILDREN. *(E.)* Davidson, G. P.; Bishop, R. F.; Townley, R. R. W.; Holmes, I. H.; Ruck, B. J. (R. Child. Hosp., Melbourne, Australia). *Lancet* 1(7901):242-245, 1975.

In a year-long survey of acute enteritis in children, a total of 378 patients were studied (209 males and 169 females, aged 10 days to 12 1/2 yr). An etiological agent was found in approximately 75% of the cases: a newly described virus belonging to the family *Reoviridae*, duovirus, was seen in children with acute enteritis but not in control children. Mixed infections of duovirus with adenovirus (10), enterovirus (7), "enteropathogenic" *E. coli* (4), or *Salmonella* species (3) were seen in 24 of the children. Acute enteritis was most common during the winter months. Duoviruses were the most common enteric pathogens during all but the 2 hottest months of the year. Duovirus infection peaked during early winter, occurring in 73% of the patients. Duovirus infection was a common cause of enteritis in each age group up to 5 yr of age. The highest percentage of infections (66%) occurred in the 6-12-month-old group. The youngest child from whom duovirus particles were recovered was 10 days old. The duration of infection was brief, with a probable incubation period of less than 2 days. No duovirus particles were seen in feces from any of 16 patients so tested after 8 days from onset of symptoms. Infections acquired in the hospital were not uncommon. Of the 25 children who developed symptoms of acute enteritis while in the hospital for another illness, 22 showed duovirus particles in stool extracts. There were 4 fatal cases of enteritis in this study. The name "duovirus" is proposed for this new virus, as opposed to "rotavirus" as has previously been suggested. It is suggested that this new virus will prove to be the most important etiological agent of sporadic acute enteritis in young children.

2330 NECROTIZING ENTEROCOLITIS. *(E.)* Frantz, III, I. D.; L'Heureux, P.; Engel, R. R.; Hunt, C. E. (Dep. Pediatr., Univ. Minnesota, Minneapolis). *J. Pediatr.* 86(2):259-263, 1975.

The diagnosis, management and clinical course of necrotizing enterocolitis was studied during a 39 month period, in 54 cases of necrotizing enterocolitis developed in a neonatal intensive care unit, which represents an incidence of 4.7% of all neonatal admissions. Of these 54 cases, 49 (91%) were premature, representing an incidence of 7.3% of all premature infants admitted during this time period. The other 5 patients included 1 infant following corrective surgery for complete transposition of the great vessels, 1 with severe respiratory distress syndrome, 1 with severe birth asphyxia, and 2 with congenital heart disease. Age at onset ranged from 1-22 days, mean 6.5 days. Roentgenographic findings showed all but 5 patients had pneumatosis intestinalis. Gas in the portal venous system occurred in 8, 2 of whom survived surgery. Pneumoperitoneum secondary to intestinal perforation occurred in 5 patients, 2 of whom survived. Of the 54 patients, 51 were evaluated for disseminated intravascular coagulation and 26 showed positive results. Surgical treatment was performed on any patient if one or more of the following were present at diagnosis: intestinal perforation, metabolic acidosis and/or shock, or disseminated intravascular coagulation. The 16 patients managed medically received continuous nasogastric decompression for at least 10 days, parenteral antibiotic therapy of gentamicin and penicillin, and i.v. alimentation until enteric feedings were reinstituted after 10 days. Of these 16, 7 (44%) survived. Of the 38 treated surgically, 12 (31%) survived. The clinical course of the 54 patients was compared to a matched group of 98 control patients. On the day of life that necrotizing enterocolitis occurred, all 54 patients and 63% of the controls were receiving standard formula feedings, both at 80 cal/kg/day. Stool cultures at that time revealed a significantly increased frequency of *Klebsiella* in necrotizing enterocolitis patients as compared to contols. It is suggested that the combined presence of certain intestinal bacteria and enteric feedings, perhaps requiring a background of mucosal ischemia, may be of etiological significance in the development of necrotizing enterocolitis and pneumatosis intestinalis.

2331 INTRAARTERIAL VASOPRESSIN IN THE TREATMENT OF UPPER GASTROINTESTINAL HEMORRHAGE: A PROSPECTIVE, CONTROLLED CLINICAL TRIAL. *(E.)* Conn, H. O.; Ramsby, G. R.; Storer, E. H.; Mutchnick, M. G.; Joshi, P. H.; Phillips, M. M.; Cohen, G. A.; Fields, G. N.; Petroski, D. (VA Hosp., West Haven, Conn.). *Gastroenterology* 68(2):211-221, 1975.

A trial was undertaken to compare conventional treatment of massive upper gastrointestinal hemorrhage with conventional therapy plus intraarterial vasopressin. Sixty episodes of upper gastrointestinal hemorrhage were evaluated in 53 patients (51 men and 2 women, aged 24-83 yr). Conventional treatment was used in 32 episodes and vasopressin was added in 28. Cirrhosis was present in 34 patients (64%). Bleeding occurred twice in 7 of the cirrhotic patients. Bleeding originated from esophageal or gastric varices in 33 episodes (55%). Vasopressin (1 U/ml) was administered at an initial rate of 0.05 to 0.20 U/min. The dose was increased until bleeding decreased. Subsequent dosage of vasopressin did not exceed 0.4 U/min. Duration of vasopressin therapy ranged from 7-74 hr (mean 28); conventional treatment, 3-64 hr (mean 22). In 9 of the conventionally treated patients (28%) and in 20 of those receiving vasopressin (71%), the hemorrhages were successfully controlled. Based on cessation of hemorrhage for at least 5 days, conventional therapy was successful in only 16% and vasopressin in only 26% of the patients. In cases of nonvariceal hemorrhage, conventional therapy was successful in

5 of 16 episodes (31%) and vasopressin in 8 of 11 (73%). For variceal hemorrhage, 4 of 16 treated conventionally (25%) and 12 of 17 receiving vasopressin (71%) had the bleeding controlled. Vasopressin therapy caused a reduction in transfusion requirements from 7.1 U/patient before to 3.7 U/patient after therapy was started. Such a drop was not noted with conventional therapy. Of the 32 conventionally treated episodes, 18 ended fatally (56%) compared to 15 of 28 treated with vasopressin (54%). There was no significant effect of either form of therapy on survival of cirrhotic versus noncirrhotic patients; or on survival of those bleeding from nonvariceal sites or from varices. Of 20 patients in whom conventional therapy failed, 10 were successfully treated with vasopressin and, of these 10, 7 survived. Complications occurred in only 3 patients treated conventionally, all had arrhythmias, 2 of whom had hypertension and bradycardia and the other had atrial fibrillation. The vasopressin group shared 18 complications among 12 patients, which included hypertension, bradycardia, other arrhythmias, cardiac arrest, respiratory arrest, and technical vascular complications. It is suggested that, if bleeding can be stopped with vasopressin, elective surgery can be performed whenever appropriate; if not, even transient control may delay action until surgery can be performed on a semi-elective basis, rather than as an emergency.

2332 IMMUNOCHEMICAL CONFIRMATION OF GASTROINTESTINAL BLEEDING. *(E.)* Adams, E. C.; Layman, K. M. (Ames Res. Lab., Elkhart, Ind.). *Ann. Clin. Lab. Sci.* 4(5):343-349, 1974.

Hemoglobin was detected by hemagglutination inhibition and by direct slide latex agglutination in fecal extracts from subjects who had gastrointestinal bleeding or who had ingested their own blood (10-20 ml as liquid or lyophilized blood in gelatin capsules). By using the direct slide agglutination test with human hemoglobin and erythrocyte stroma antigens, it was possible to determine the approximate site of bleeding. Fecal extracts from patients with bleeding from the duodenum or above were positive for hemoglobin but not stroma, while those from patients with bleeding from the lower part of the colon were positive for both hemoglobin and stroma. Fecal extracts from patients who ingested their own blood became positive for hemoglobin 10 hr after ingestion and remained positive for several days. The stroma test was negative after ingestion of blood by either means. Neither of the tests for human hemoglobin nor the test for stroma was positive on fecal extracts from subjects on a meat diet, although the peroxidase test was strongly positive.

2333 THE EFFECT OF CHOLESTYRAMINE ON THE ELIMINATION OF CHOLESTEROL AS BILE ACIDS IN PATIENTS WITH HYPERLIPOPROTEINAEMIA TYPE II and IV. *(E.)* Einarsson, K.; Hellstrom, K.; Kallner, M. (Serafim Hosp., Stockholm, Sweden). *Eur. J. Clin. Invest.* 4(6):405-410, 1974.

The turnover of ^{14}C-cholic acid and ^{3}H-chenodeoxycholic acid was studied in 10 hyperlipemic patients (5 men and 5 women, aged 45-61 yr) before and during treatment with cholestyramine (10-12 g/day). In the 5 female patients with hyperlipoproteinemia type IIa the pool size prior to treatment averaged 320 mg and the turnover of cholic acid averaged 96 mg/day. Corresponding values for chenodeoxycholic acid were 728 mg and 168 mg/day. During cholestyramine treatment, cholic acid snythesis rose 4 to 18-fold and the turnover of chenodeoxycholic acid increased to a mean of 379 mg/day. Combined pool size of cholic acid and chenodeoxycholic acid averaged 1048 mg before and 1389 mg during treatment with cholestyramine. Values for total bile acid formation were 264 and 1116 mg/day, resp. In 3 of the patients, the changes due to therapy were associated with a normalization of the serum cholesterol levels. Due primarily to the high turnover of cholic acid, total bile acid synthesis was above normal in the 5 male patients with hyperlipoproteinemia type IV. During cholestyramine treatment, the serum lipids and cholic acid formation were unchanged, but the turnover of chenodeoxycholic acid increased by a factor of 2. This caused a decrease of the abnormally high initial ratio between the formation of cholic acid and chenodeoxycholic acid from 3.3 to 1.5. The unequal response to cholestyramine treatment is taken to be a further indication that hyperlipoproteinemia type II and type IV are different entities of disease affecting cholesterol and bile acid metabolism. It is not certain at this time whether the characteristic pattern encountered for bile acid formation in patients with hyperlipoproteinemia types II and IV is due to differences in the catabolic pathways of cholesterol.

2334 EOSINOPHILIC INFILTRATION OF DUODENUM AND PANCREATIC HEAD: REPORT OF A CASE STUDIED ARTERIOGRAPHICALLY. *(E.)* Ryan, K. G. (Woodland Clin. Med. Group, Calif.). *Surgery* 77(2):321-324, 1975.

A 39 yr old man presented with a 2 week history of right upper quandrant abdominal pain, nausea, vomiting, and intermittent chills and fever. An i.v. cholangiogram was normal. All other studies were negative except for a slightly elevated serum alkaline phosphatase. Six months later the pain returned in the epigastrium and right upper quadrant followed by 2 or 3 episodes of bright red blood in the stools. A lesion involving the second portion of the duodenum was thought to be carcinoma of the head of the pancreas. One month later a barium study of the duodenum showed a segment in the descending duodenum which remained persistently narrow with a predominant impression on the lateral aspect, interpreted as a lesion either intrinsic to the duodenum or circumferential about the duodenum. Pancreatic arteriography was carried out with selective celiac axis and superior mesenteric artery catheterization. Due to the absence of arteriographic abnormality and the appearance on the duodenogram, the radiological diagnosis favored a benign intrinsic duodenal lesion, such as scarring from peptic ulcer or regional enteritis, with the possibility that this could represent annular pancreas. Surgery revealed a mass involving the head of the pancreas and duodenum, diagnosed surgically as

carcinoma of the head of the pancreas, and radical pancreatoduodenectomy, cholecystectomy, antrectomy, and vagotomy were carried out. Pathological examination showed no evidence of malignancy or of parasitic infestation. Pathological diagnosis was eosinophilic duodenitis and pancreatitis with inflammatory pseudotumor. Few reports of this disease show lesions confined to the duodenum. It is also uncommon to see involvement of adjacent organs other than regional lymph nodes. It is noted that the entirely normal angiographic findings would be very unusual if the mass demonstrated on the barium studies were indeed carcinoma. A normal vascular pattern arteriographically demonstrated in the mass is suggested as a preoperative clue to a benign diagnosis which should raise the possibility of eosinophilic granuloma. Conservative management is recommended for this disease.

2335 PAROTID LYMPHANGIOMA: A CONGENITAL TUMOUR. *(E.)* Kornblut, A. D.; Ilse, H.; Haubrich, J. (HNO Clin., Univ. Gottingen, Germany). *Otorinolaringol. Oftalmol.* 35(5):303-314, 1973.

2336 CYSTIC ADENOMA OF A MINOR SALIVARY GLAND: A HISTOCHEMICAL STUDY. *(E.)* Harrison, J. D. (King's Coll. Hosp. Dent. Sch., London, England). *J. Pathol.* 114(1):29-38, 1974.

2337 LIGHT CHAIN TYPE RATION OF IgA-PRODUCING CELLS IN HUMAN JEJUNAL BIOPSIES. *(E.)* Mul, N. A. J.; de Boer, L. E. M.; Taminiau, J. A.; Ballieux, R. E. (Univ. Hosp., Utrecht, Netherlands). *Eur. J. Clin. Invest.* 5(1):63-67, 1975.

2338 LYMPHOGENIC ENTEROPATHY FROM THORACIC DUCT OBSTRUCTION. *(E.)* Molnar, Z.; Karoliny, G.; Mozsik, G.; Nemeth, A. (Univ. Med. Sch., Pecs, Hungary). *Lymphology* 7(3):130-136, 1974.

2339 ANTIBIOTICS AND FAECAL TRYPTIC ACTIVITY. *(E.)* Morrison, P. S.; Barnes, G. L. (St. Bartholemew's Hosp., London, England). *NZ Med. J.* 80(530):544-545, 1974.

2340 BEHAVIORAL EFFECTS OF DIETARY GALACTOSE IN GALACTOSEMIA HETEROZYGOTES. *(E.)* Frazier, P. D. M.; Frazier, J. R.; Summer, G. K. (Univ. North Carolina Sch. Med., Chapel Hill). *Fed. Proc.* 34(3):930, 1975.

2341 IMPAIRMENT OF UPPER ALIMENTARY VAGAL FUNCTION IN DIABETES. *(E.)* Stewart, I. M.; Hosking, D. J.; Preston, B. J. Atkinson, M. (Nottingham Gen. Hosp., England). *Gut* 15(10):832, 1974.

2342 DEFICIENCY OF LIPOLYTIC PANCREATIC ENZYMES IN AN ADULT MAN. *(E.)* Arnesjo, B.; Ihse, I.; Odeberg, H.; Stahl, E. (Univ. Hosp. Lund, Sweden). *Digestion* 11(1/2):147-151, 1974.

2343 SURGICAL LAPAROSCOPY. *(Rus.)* Prudkov, I. D. (Sverdlovsk Med. Inst., USSR). *Klin. Khir.* (9):56-60, 1974.

2344 THE SECRETIN PROVOCATION TEST IN THE DIAGNOSIS OF ZOLLINGER-ELLISON SYNDROME. *(E.)* Bonfils, S.; Mignon, M.; Accary, J. P. (INSERM, Bichat Hosp., Paris, France). *Gut* 15(10):841, 1974.

2345 THE EFFECT OF MAGNESIUM OF GASTRIN LEVELS IN DUODENAL ULCER AND ZOLLINGER-ELLISON PATIENTS. *(E.)* Fender, H. R.; Thompson, J. C. (Univ. Texas Med. Branch, Galveston). *Fed. Proc.* 34(3):442, 1975.

2346 EFFECT OF UROGASTRONE IN THE ZOLLINGER-ELLISON SYNDROME. *(E.)* Elder, J. B.; Ganguli, P. C.; Gillespie, I. E.; Delamore, I.; Gregory, H. (R. Infirm., Manchester, England). *Gut* 15(10):840, 1974.

2347 ABDOMINAL PREGNANCY AS A CAUSE OF FRANK MELENA. *(E.)* Machudo, A. L.; de Menezes, D. B.; Rabelo, J. I. C. (Gen. Hosp., Fortaleza, Brazil). *Arq. Gastroenterol.* 11(4):237-240, 1974.

2348 PROTEIN-CALORIE MALNUTRITION IN INFANT NON-HUMAN PRIMATES: CORRELATION OF BIOCHEMICAL AND ULTRASTRUCTURAL ALTERATIONS IN THE LIVER. *(E.)* Worthington, B. S.; Enwonwu, C. O. (no affil.). *Fed. Proc.* 34(3):941, 1975.

2349 ASSESSMENT OF STEATORRHOEA IN MALNOURISHED CHILDREN. *(E.)* Gracey, M.; Thomas, J.; Suharjono; Hendardji, H. (Princess Margaret Child. Med. Res. Found., Perth, Australia). *Environ. Child Health* 20(5):223-225, 1974.

2350 PLATELET AND FIBRINOGEN SURVIVAL IN KALA-AZAR. *(E.)* Musumeci, S.; D'Agata, A.; Panebianco, M. G. (Dep. Pediatr., Univ. Catania, Italy). *Trans. R. Soc. Trop. Med. Hyg.* 68(5):360-367, 1974.

2351 CHARACTERIZATION OF VIBRIOS, ISOLATED AT VELLORE, AND CORRELATION WITH EPIDEMIOLOGIC PATTERNS OF CHOLERA. *(E.)* Koshi, G.; Brahmadathan, K. N. (Christ. Med. Coll. Hosp., Vellore, India). *Indian J. Med. Res.* 61(12):1730-1736, 1973.

2352 POSSIBLE ROLE OF ANIMAL FEEDING-STUFFS IN SPREAD OF ANTIBIOTIC-RESISTANT INTESTINAL COLIFORMS. *(E.)* Howells, C. H. L.; Joynson, D. H. M. (Univ. Hosp. Wales, Cardiff). *Lancet* 2(7899):156-157, 1975.

2353 REDUCTION OF GASTROENTERITIS MORBIDITY IN HIGH-RISK INFANTS. *(E.)* Nutting, P. A.; Strotz, C. R.; Shorr, G. I.; Berg, L. E. (Indian Health Serv., Tucson, Ariz.). *Pediatrics* 55(3):354-358, 1975.

2354 DIAGNOSIS AND TREATMENT OF INFECTIOUS ENTERITIS. *(Ger.)* Blum, A. L. (Triemli Munic. Hosp., Zurich, Switzerland). *Therapiewoche* 24(9):933-941, 1974.

2355 ASSOCIATION OF Salmonella typhimurium WITH, AND ITS INVASION OF, THE ILEAL MUCOSA IN MICE. *(E.)* Tannock, G. W.; Blumershine, R. V. H.; Savage, D. C. (Dep. Microbiol., Univ. Illinois, Urbana). *Infect. Immun.* 11(2):365-371, 1975.

2356 DISACCHARIDE HYDROLYSIS, INTESTINAL ABSORPTION AND ELECTROGENIC PROPERTIES IN Salmonella ENTEROCOLITIS IN MICE. *(E.)* Madge, D. S. (Dep. Biol. Sci., Wye Coll., England). *Digestion* 11(1/2):25-38, 1974.

2357 FAECAL CARRIAGE OF Staphylococcus aureus IN INFANTILE ENTERITIS DUE TO ENTEROPATHIC Escherichia coli. *(E.)* Hone, R.; Keane, C. T.; Fitzpatrick, S. (Adelaide Hosp., Dublin Univ., Ireland). *Scand. J. Infect. Dis.* 6(4):329-332, 1974.

2358 COMPARATIVE STUDY OF EPIDEMIOLOGICAL FEATURES AND THE CLINICAL COURSE OF ENTERITIS CAUSED BY ENTEROPATHOGENIC Escherichia coli 0124 AND CRIMEA. *(Rus.)* Milovanova, L. P.; Bondarenko, V. I.; Kubasov, I. G.; Bellavina, G. S.; Gallinskaia, S. M.; Gertsryken, S. L. (Kiev Inst. Epidemiol. Microbiol. Parasitol., USSR). *Zh. Mikrobiol. Epidemiol. Immunobiol.* (6):75-78, 1974.

2359 SELECTION RATE OF DRUG-RESISTANT FORMS OF Escherichia AS A FUNCTION OF THE ANTIBIOTICS USED TO TREAT PATIENTS WITH ACUTE DYSENTERY. *(Rus.)* Astapov, A. A.; Amvrosiev, Iu. A.; Tsynkalovskii, I. B. (Kuban Med. Inst., Stavropol, USSR). *Antibiotiki* 19(3):279, 1974.

2360 AGGLUTINATING ANTIBODIES IN THE DUODENAL SECRETIONS OF INFANTS WITH ENTEROPATHIC E. coli GASTROENTERITIS. *(E.)* McNeish, A. S.; Gaze, H.; Evans, N. (Inst. Child Health, Birmingham, England). *Gut* 15(10):834, 1974.

2361 SOME SURGICAL MANIFESTATIONS OF MESENTERIAL LYMPHADENITIS, ASSOCIATED WITH INFECTION OF THE Yersinia enterocolitica. *(E.)* Saebo, A. (Akershus Gen. Hosp., Nordbyhagen, Norway). *Acta Chir. Scand.* 140(8):655-657, 1974.

2362 ANALYSIS OF UNIDIRECTIONAL FLUXES OF SODIUM DURING DIARRHEA INDUCED BY Clostridium perfringens ENTEROTOXIN IN THE RAT TERMINAL ILEUM. *(E.)* McDonel, J. L.; Asano, T. (Dep. Microbiol., Univ. Notre Dame, Indiana). *Infect. Immun.* 11(3):526-529, 1975.

2363 INTESTINAL ION FLUX IN TRANSMISSIBLE GASTROENTERITIS (TGE). *(E.)* McClung, H. J.; Butler, D. G.; Hamilton, J. R. (Hosp. Sick Child., Toronto, Canada). *Fed. Proc.* 34(3):916, 1975.

2364 SHIGA BACILLUS DYSENTERY ASSOCIATED WITH MARKED LEUKOCYTOSIS AND ERYTHROCYTE FRAGMENTATION. *(E.)* Mujibur Rahaman, M.; Jamiul Alam, A. K. M.; Islam, M. R.; Greenough III, W. B.; Lindenbaum, J. (Cholera Res. Lab., Dacca, Bangladesh). *Johns Hopkins Med. J.* 36(2):65-70, 1975.

2365 CLINICAL AND LAPAROTOMY PARALLELS IN ACUTE DYSENTERY IN ADULTS. *(Rus.)* Evdokimova, T. V.; Podlevskii, A. F.; Iafaev, R. Kh. (Leningrad Med. Inst. Sanit. Hyg., USSR). *Zh. Mikrobiol. Epidemiol. Immunobiol.* (6):82-85, 1974.

2366 SMALL INTESTINAL MUCOSAL MORPHOLOGY IN A GROUP OF INFANTS AND YOUNG CHILDREN WITH DELAYED RECOVERY AFTER ACUTE DIARRHOEA AND VOMITING. *(E.)* Boyce, M. J.; France, N. E.; Walker-Smith, J. A. (no affil.). *Gut* 15(10):827, 1974.

2367 LOPERAMIDE (R 18 553), A NOVEL TYPE OF ANTIDIARRHEAL AGENT. PART 1: IN VIVO ORAL PHARMACOLOGY AND ACUTE TOXICITY. COMPARISON WITH MORPHINE, CODEINE, DIPHENOXYLATE AND DIFENOXINE. *(E.)* Niemegeers, C. J. E.; Lenaerts, F. M.; Janssen, P. A. J. (Res. Lab., Janssen Pharm., Beerse, Belgium). *Arzneim. Forsch.* 24(10):1633-1666, 1974.

2368 LOPERAMIDE (R 18 553), A NOVEL TYPE OF ANTIDIARRHEAL AGENT. PART 2: IN VIVO PARENTERAL PHARMACOLOGY AND ACUTE TOXICITY IN MICE. COMPARISON WITH MORPHINE, CODEINE AND DIPHENOXYLATE. *(E.)* Niemegeers, C. J. E.; Lenaerts, F. M.; Janssen, P. A. J. (Res. Lab., Janssen Pharm., Beerse, Belgium). *Arzneim. Forsch.* 24(10):1636-1640, 1974.

2369 LOPERAMIDE (R 18 553), A NOVEL TYPE OF ANTIDIARRHEAL AGENT. PART 3: IN VITRO STUDIES ON THE PERISTALTIC REFLEX AND OTHER EXPERIMENTS ON ISOLATED TISSUES. *(E.)* Van Nueten, J. M.; Janssen, P. A. J.; Fontaine, J. (Res. Lab., Janssen Pharm., Beerse, Belgium). *Arzneim. Forsch.* 24(10):1641-1645, 1974.

2370 LOPERAMIDE (R 18 553), A NOVEL TYPE OF ANTIDIARRHEAL AGENT. PART 4: STUDIES ON SUBACUTE AND CHRONIC TOXICITY AND THE EFFECT ON REPRODUCTIVE PROCESSES IN RATS, DOGS AND RABBITS. *(E.)* Marsboom, R.; Herin, V.; Verstraeten, A.; Vandesteene, R.; Fransen, J. (Res. Lab., Janssen Pharm., Beerse, Belgium). *Arzneim. Forsch.* 24(10):1645-1649, 1974.

2371 LOPERAMIDE (R 18 553), A NOVEL TYPE OF ANTIDIARRHEAL AGENT. PART 5: THE PHARMACOKINETICS OF LOPERAMIDE IN RATS AND MAN. *(E.)* Heykants, J.; Michiels, M.; Knaeps, A.; Brugmans, J. (Dep. Drug Metab., Janssen Pharm., Beerse, Belgium). *Arzneim. Forsch.* 24(10):1649-1653, 1974.

2372 LOPERAMIDE (R18 553), A NOVEL TYPE OF ANTIDIARRHEAL AGENT. PART 6: CLINICAL PHARMACOLOGY. PLACEBO-CONTROLLED COMPARISON OF THE CONSTIPATING ACTIVITY AND SAFETY OF LOPERAMIDE, DIPHENOXYLATE AND CODEINE IN NORMAL VOLUNTEERS. *(E.)* Schuermans, V.; Van Lommel, R.; Dom, J.; Brugmans, J. (Res. Lab., Janssen Pharm., Beerse, Belgium). *Arzneim. Forsch.* 24(10):1653-1657, 1974.

2373 LOPERAMIDE (R 18 553), A NOVEL TYPE OF ANTIDIARRHEAL AGENT. PART 7: CLINICAL INVESTIGATION. EFFICACY AND SAFETY OF LOPERAMIDE IN PATIENTS WITH SEVERE CHRONIC DIARRHEA. *(E.)* Verhaegen, H.; De Cree, J.; Schuermans, V. (St. Bartholomeus Hosp., Merksem-Antwerp, Belgium). *Arzneim. Forsch.* 24(10):1657-1660, 1974.

2374 LOPERAMIDE (R 18 553), A NOVEL TYPE OF ANTIDIARRHEAL AGENT. PART 8: CLINICAL INVESTIGATION. USE OF A FLEXIBLE DOSAGE SCHEDULE IN A DOUBLE-BLIND COMPARISON OF LOPERAMIDE WITH DIPHENOXYLATE IN 614 PATIENTS SUFFERING FROM ACUTE DIARRHEA. *(E.)* Dom, J.; Leyman, R.; Schuermans, V.; Brugmans, J. (Dep. Clin. Res., Janssen Pharm., Beerse, Belgium). *Arzneim. Forsch.* 24(10):1660-1665, 1974.

2375 A MULTICENTRE DOUBLE-BLIND STUDY IN ACUTE DIARRHOEA COMPARING LOPERAMIDE (R 18 553) WITH TWO COMMON ANTIDIARRHOEAL AGENTS AND A PLACEBO. *(E.)* Amery, W.; Duyck, F.; Polak, J.; Ven Den Bouwhuysen, G. (Res. Lab., Janssen Pharm., Beerse, Belgium). *Curr. Ther. Res.* 17(3):263-270, 1975.

See also, 1598, 1727, 1846, 2013, 2051, 2074, 2379.

GENERAL

Parasitology

2376 SERUM IgE LEVELS IN PROTOZOAL AND HELMINTHIC INFECTIONS. *(E.)* Radermecker, M.; Bekhti, A.; Poncelet, E.; Salmon, J. (Hosp. Baviere, Univ. Liege, Belgium). *Int. Arch. Allergy Appl. Immunol.* 47(2):285-295, 1974.

Serum IgE concentration was measured according to the Rowe modification of the Mancini technique in non-atrophic patients suffering from various helminthic or protozoal infections. Normal serum IgE levels ranged from 100-370 U/ml. An increment of IgE levels above the 95% confidence limits was noted in 67% of the 40 patients with systemic parasitosis and in 37% of 8 patients with protozoal infections. All but one of 23 patients with intestinal parasitosis had normal serum IgE concentration. High serum IgE levels were regularly found in schistosomiasis, strongyloidiasis, echinococciasis, filariasis and hookworm disease. All 17 patients with threadworms or tapeworms had normal IgE levels. In 6 patients, the serum IgE levels were followed periodically before and after specific treatment of the parasitosis. In 2 cases of schistosomiasis, after niridazole administration (25 mg/kg for 7 days) the IgE values rose sharply immediately following onset of therapy. The values decreased slowly. Similar results were noted in 1 of 2 patients receiving thiabendazole (25 mg/kg for 1 day) for strongyloidiasis. These results suggest that the capacity of helminthic antigens to potentiate IgE synthesis is related to the parasite's ability to migrate into host tissues. These data also show a reduction in IgE level following specific treatment of strongyloidiasis, schistosomiasis, and amebiasis. Additionally, they show that an effective anti-parasitic treatment may produce a rapid and sometimes considerable (10 times) increase of serum IgE levels even though it was within normal limits. This boosting effect of antihelminthic treatment is likely to originate in the abrupt release of parasitic antigens from the killed parasites. It is suggested that repetitive quantitation of IgE from serum in patients with vague clinical pictures, suggestive of parasitic infestation, may be of some diagnostic value. The sharp increase in IgE levels is likely during the fourth week following administration of an effective treatment and a significant reduction is expected 5 months later.

2377 COMPARISON OF PYRANTEL AND MEBENDAZOLE IN THE TREATMENT OF ANCYLOSTOMIASIS: STUDY OF 40 CASES. *(Por.)* Conte, V. P.; Zaterka, S.; Costa, F. A. B.; Bettarello, A. (Fac. Med., Univ. Sao Paulo, Brazil). *Arq. Gastroenterol. Sao Paulo* 11(3):143-149, 1974.

Of 40 patients with ancylostomiasis and necatoriasis, either alone or in association with other parasitic diseases, 20 (12 males and 8 females, including 3 children) received pyrantel (20 mg/kg/day p.o. on 2 consecutive days) and 20 (9 males and 11 females, including 8 children) received mebendazole (200 mg/day on 3 consecutive days). Patients in both groups lived in an inland farm and were studied for 90 days after treatment. Parasitological examination of the stools showed that pyrantel cured 9 patients and reduced the number of eggs/g stools by an average of 73.3% in the remaining 11. Ascariasis was cured in all 5 patients in whom it was diagnosed, while 8 of

10 patients with whipworm responded to treatment. Mild side effects, which developed in 6 patients, consisted of anorexia, epigastric and abdominal pain, vertigo, pruritus, and diarrhea. Mebendazole cured 10 of the 20 patients who received it and reduced the number of eggs/g stool by an average of 86.3% in the remaining 10. All of 8 patients with associated ascariasis responded to mebendazole, as did 9 of the 11 with whipworm. Side effects, which occurred in 4 patients, consisted of vertigo, abdominal pain, and diarrhea. These results confirm that both pyrantel and mebendazole are effective in the treatment of hookworm and are well tolerated by most patients.

2378 DIFFERENCES IN IMMUNITY TO Nematospiroides dubius IN INBRED AND OUTBRED MICE. *(E.)* Lueker, D. C.; Hepler, D. I. (Dep. Microbiol., Colorado State Univ., Fort Collins). *J. Parasitol.* 61(1):159-160, 1975.

2379 THE TRANSMISSION OF HOOKWORMS, Salmonella, AND Shigella BY PALM WINE. *(Ger.)* Okafor, N.; Ikeme, M. M. (Dept. Microbiol., Univ. Nigeria, Nsukka). *Arch. Lebensmittelhyg.* 26(1):37-38, 1975.

2380 TREATMENT OF PERFORATED GASTRIC AND DUODENAL ULCERS IN PATIENTS WITH OPISTHORCHIASIS. *(Rus.)* Kirilenko, M. P.; Pinekenshtein, A. A. (Tyumen Reg. Clin. Hosp., USSR). *Klin. Khir.* (9):61-62, 1974.

2381 VERMINOUS APPENDICITIS CAUSED BY Enterobius vermicularis. *(Sp.)* Jofre Gutierrez, J. A.; Tersoglio, A. E.; Serra, D. E.; Gomez, S. C. (Fac. Med. Sci., Natl. Univ. Cuyo, Mendoza, Argentina). *Rev. Esp. Enferm. Apar. Dig.* 44(6):575-580, 1974.

2382 ANTIAMOEBIC ACTION OF ETOPHAMIDE IN CHILDREN. *(E.)* Biagi, F.; Alvarez, R.; Gonzalez, C. (no affil.). *Trans. R. Soc. Trop. Med. Hyg.* 68(5): 368-369, 1974.

2383 INTER-AND INTRAFAMILIAL DISTRIBUTION OF Giardia lamblia INFECTION. *(E.)* Kettis, A. A.; Thoren, G. (Hosp. Infect. Dis., Stockholm, Sweden). *Scand. J. Infect. Dis.* 6(4):349-353, 1974.

2384 STUDIES ON MEDITERRANEAN LEISHMANIASIS. I. AN OUTBREAK OF VISCERAL LEISHMANIASIS IN NORTHERN ITALY. *(E.)* Pampiglione, S.; La Place, M.; Schlick, G. (Fac. Vet. Med., Univ. Bologna, Italy). *Trans. R. Soc. Trop. Med. Hyg.* 68(5):349-359, 1974.

2385 ANTHELMINTIC EFFECT OF THE GENTAMICIN COMPLEX AND COPRODUCED ANTIBIOTICS AGAINST TAPEWORMS OF LAMBS AND CATS. *(E.)* Panitz, E. (Anim. Health Res. Cent., Schering Corp., Allentown, N.J.). *J. Parasitol.* 61(1):157-158, 1975.

2386 EFFECTS OF INFECTION WITH PLEROCERCOIDS OF THE TAPEWORM, Spirometra mansonoides ON SERUM LIPIDS OF THE RAT. *(E.)* Phares, C. K.; Carroll, R. M.; Ruegamer, W. R. (Univ. Nebraska Med. Cent., Omaha). *Fed. Proc.* 34(3):939, 1975.

See also, 1923, 2182, 2189, 2210, 2211, 2264, 2316.

GENERAL

Regional Enteritis

2387 THE PULMONARY EXCRETION OF $^{14}CO_2$ IN PATIENTS WITH ILEAL RESECTION, CROHN'S DISEASE AND CONTROL SUBJECTS FOLLOWING ADMINISTRATION OF GLYCINE-1-^{14}C. *(E.)* Belfield, A.; Melikian, V.; Rao, K. P.; Stokes, P. O.; Cooke, W. T. (Gen. Hosp., Birmingham, England). *Experientia* 30(12):1429-1430, 1974.

Glycine-1-^{14}C (1 ml of glycine solution added to water) was administered immediately before breakfast to patients with bile salt deconjugation and to a control group with normal ileal function. The excretion of $^{14}CO_2$ in the breath was measured at 1/2, 1, 1 1/2, 2, 3, 4, 6, and 8 hr after ingestion of the test dose. For patients with ileal resection the fraction of the original dose excreted in the breath in the 0-8 hr period ranged from 0.116 to 0.213, a statistically significant difference from the range of 0.153 to 0.262 observed in controls. In both groups of subjects the pulmonary excretion of $^{14}CO_2$ obeyed bi-exponential kinetics. Three of the patients with ileal resection had low serum pyridoxal phosphate concentrations and 2 subnormal serum folates. Partial metabolic blocks due to deficiency of dietary co-factors may be the cause of the reduction in the amount of glycine metabolized to carbon dioxide. In spite of the finding that a reduction in the pulmonary excretion of $^{14}CO_2$ occurs in patients with Crohn's disease and ileal resection, the measurement of $^{14}CO_2$ excretion following oral glycine-1-^{14}C administration is seen as a worthwhile preliminary to the bile salt deconjugation test. An erroneous impression of the extent of intestinal bile salt deconjugation may be obtained if there is no prior knowledge of the patient's ability to convert glycine-1-^{14}C to $^{14}CO_2$.

2388 SURGICAL MANAGEMENT OF INFLAMMATORY BOWEL DISEASE IN CHILDREN. *(E.)* Becker, J. M.; Schneider, K. M.; Krasna, I. H. (Long Island Jewish-Hillside Med. Cent., New Hyde Park, N.Y.). *Paediatrician* 3(3/5):194-203, 1974.

2389 THE RELATIONSHIP AND EPIDEMIOLOGY OF ACUTE TERMINAL ILEITIS AND CROHN'S DISEASE. *(E.)* Kewenter, J.; Hulten, L.; Kock, N. G. (Dep. Surg. II, Univ. Goteborg, Sweden). *Gut* 15(10):801-804, 1974.

2390 THE PROTEAN MANIFESTATIONS OF CROHN'S DISEASE. *(E.)* Farmer, R. G. (Cleveland Clin. Found., Ohio). *Postgrad. Med.* 57(1):129-140, 1975.

2391 CROHN'S DISEASE OF THE COLON. I. FISTULA IN CROHN'S DISEASE OF THE COLON, CLASSIFICATION PRESENTING FEATURES AND MANAGEMENT IN 63 PATIENTS. *(E.)* Greenstein, A. J.; Kark, A. E.; Dreiling, D. A. (Mt. Sinai Sch. Med., City Univ. New York, N.Y.). *Am. J. Gastroenterol.* 62(5):419-429, 1974.

2392 DIFFUSE JEJUNO-ILEITIS OF CROHN'S DISEASE. *(E.)* Cooke, W. T.; Swan, C. H. J. (Gen. Hosp., Birmingham, England). *Q. J. Med.* 43(172): 583-601, 1974.

2393 GRANULOMATOUS CHEILITIS IN CROHN'S DISEASE. *(E.)* Carr, D. (R. Victoria Infirm., Newcastle upon Tyne, England). *Br. Med. J.* 4(5945): 636, 1974.

2394 CARCINOMA OF THE SMALL INTESTINE ASSOCIATED WITH TRANSMURAL ILEITIS (CROHN'S DISEASE). *(E.)* Savage, R. A.; Farmer, R. G.; Hawk, W. A. (Cleveland Clin. Found., Ohio). *Am. J. Clin. Pathol.* 63(2):168-178, 1975.

2395 CAUSES AND TREATMENT OF DIARRHEA IN CHRONIC INFLAMMATORY BOWEL DISEASE. *(Ger.)* Eckardt, V. (1st Med. Clin., Johannes Gutenberg Univ., Mainz, Germany). *Therapiewoche* 24(22):2504-2513, 1974.

2396 PROBLEMS IN CROHN'S DISEASE: EVALUATION OF THE RESULTS OF SURGICAL TREATMENTS. *(E.)* Janowitz, H. D. (Mt. Sinai Sch. Med., City Univ. New York, N.Y.). *J. Chron. Dis.* 28(2):63-66, 1975.

2397 PROGNOSIS AFTER COLONIC RESECTION FOR CROHN'S DISEASE OF THE COLON. *(E.)* Nugent, F. W.; Veidenheimer, M. C.; Meissner, W. A.; Haggitt, R. C. (Lahey Clin. Found., Boston, Mass.). *Lahey Clin. Found. Bull.* 24(1):1-7, 1975.

2398 THE PROXIMAL JEJUNAL MUCOSA IN COLONIC CROHN'S DISEASE AND ULCERATIVE COLITIS. *(E.)* Ferguson, R.; Allan, R. N.; Cooke, W. T. (Gen. Hosp., Birmingham, England). *Gut* 15(10):839, 1974.

2399 BIDIRECTIONAL SODIUM FLUX ACROSS THE INTESTINAL MUCOSA IN CROHN'S DISEASE. *(E.)* Allan, R. N.; Steinberg, D. M.; Dixon, K.; Cooke, W. T. (Gen. Hosp., Birmingham, England). *Gut* 15(10): 839-840, 1974.

See also, 2088, 2132, 2133, 2134, 2135, 2143, 2337.

NUMBERS FOLLOWED BY AN ASTERISK INDICATE ABSTRACTS. OTHER NUMBERS REFER TO CITATIONS.

ABE, H.
2218*
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1892
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1867
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1934
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2394
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1963
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